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JANUARY 1959

DISEASES of the CHEST

OFFICIAL PUBLICATION

Silver Anniversary Year

25th Annual Meeting
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Homecoming Meeting
Albuquerque, New Mexico, October 14-17, 1959

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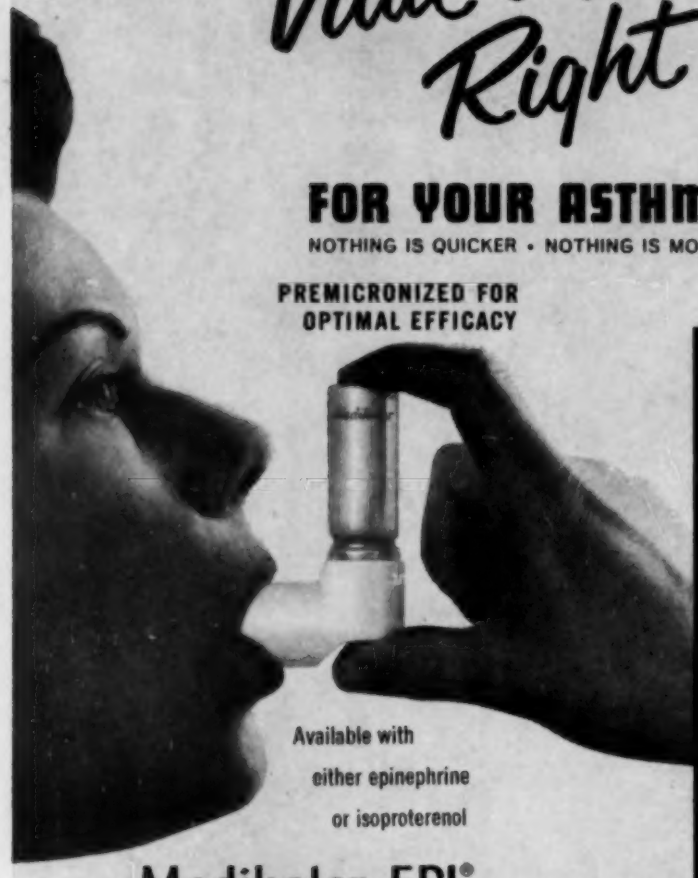

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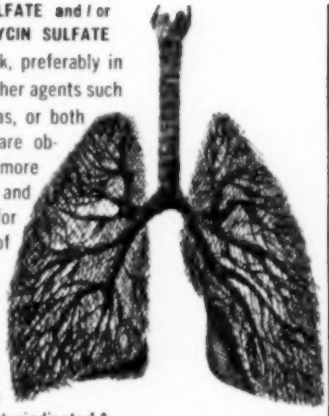

*Committee on Chemotherapy and Antibiotics, American College of Chest Physicians. Present status of chemotherapy in tuberculosis, J. Indiana M. A. 51:799 (June) 1958

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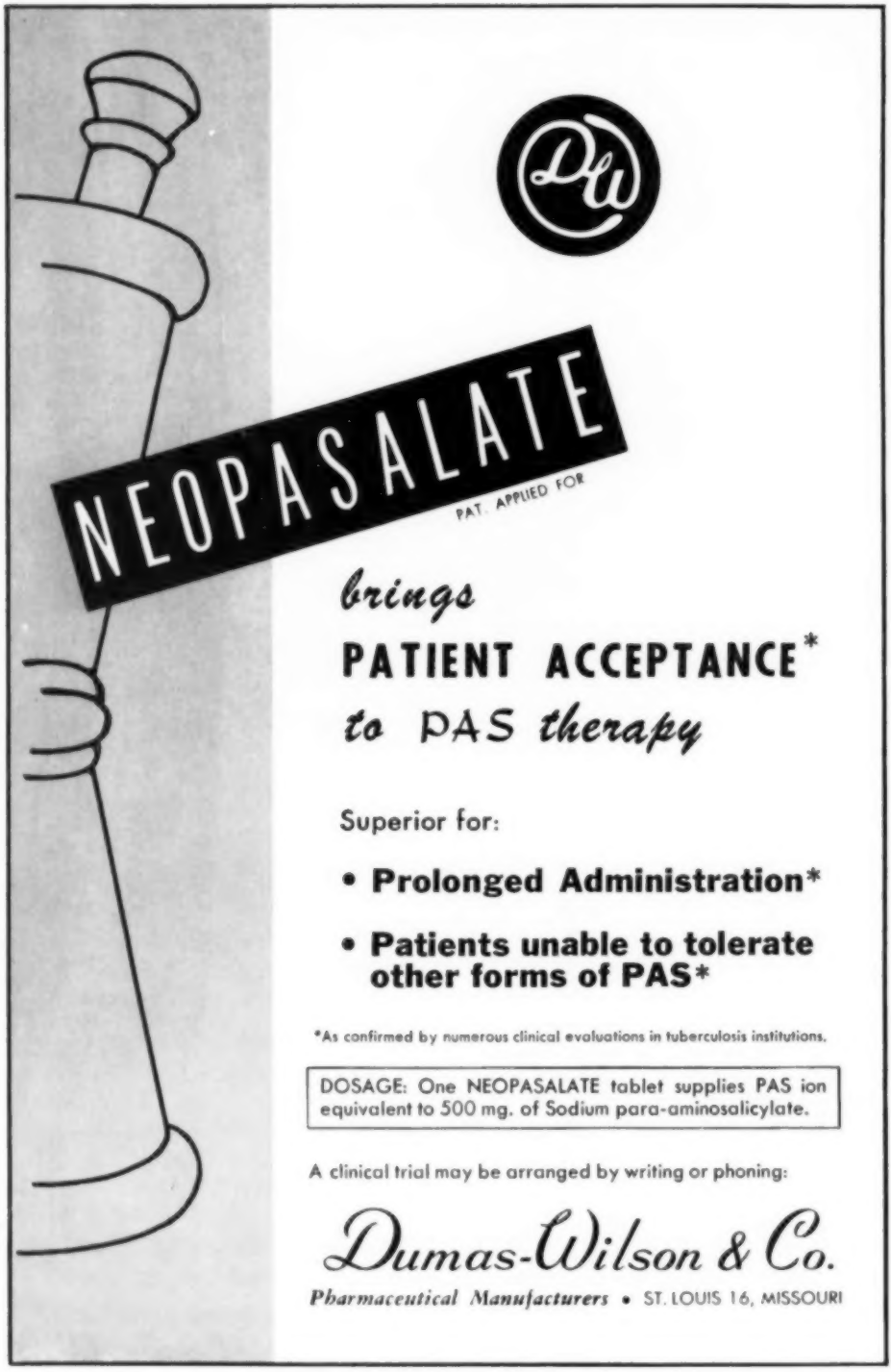
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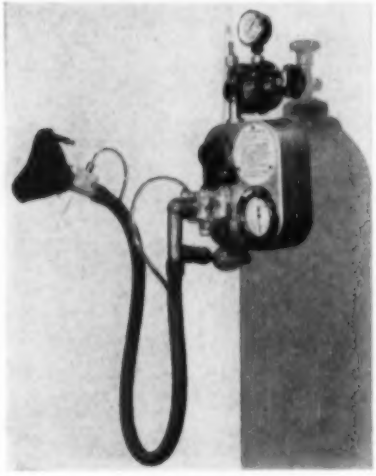

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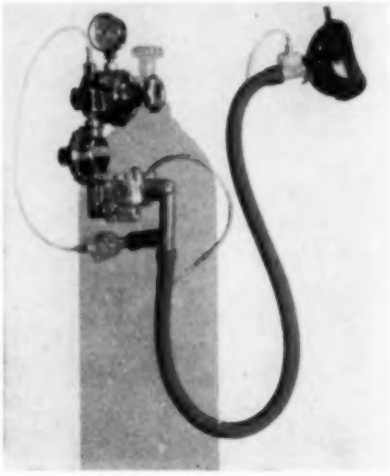

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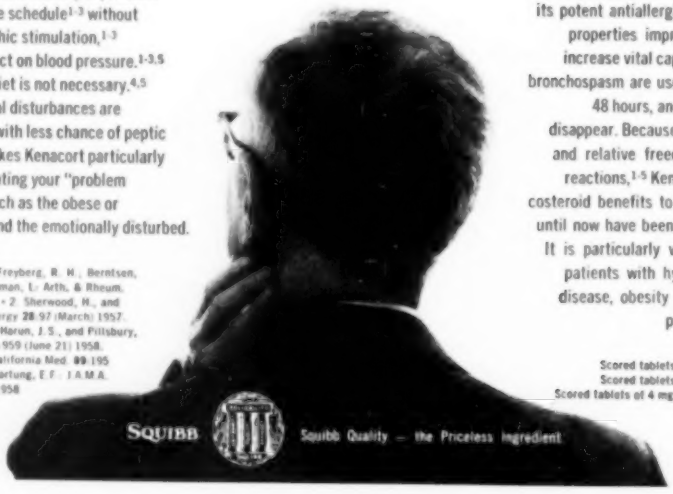

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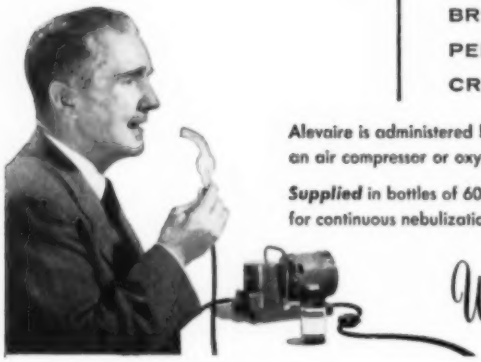

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1. Nussbaum, H. E., Leff, W. A., Mattia, V. D., Jr. and Hillman, E.: An effective combination in the treatment of the hypertensive patient. Am. J. M. Sc. **234**: 150, Aug. 1957.

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2. TESSALON acts centrally, to control cough at the level of the "cough center" in the medulla.
3. TESSALON is reported to thin sputum.[1]
4. TESSALON increases vital capacity and ventilation.
5. TESSALON improves exercise tolerance.
6. TESSALON relieves dyspnea.

Fewer coughs per minute

Shane and co-workers,[2] using the method of Bickerman and Barach,[3] induced measurable cough in 20 volunteers, using a 15 per cent citric acid aerosol as the cough-producing agent. The antitussive efficacy of TESSALON (100 mg.) was estimated to be 2½ times that of codeine (½ grain) in this test.

Cough suppressing activity of TESSALON[2]

	Average Number of Coughs*
No therapy	8.3
Codeine	4.4
TESSALON	1.7

*Based on 5-minute interval immediately following inhalation of citric acid to induce cough. Each patient was tested on three separate occasions.

Controls cough in the chest

It has been shown that the increased sensitivity of the sensory receptors in the lung during inspiration is an important part of the cough mechanism. TESSALON has a selective inhibiting effect on these dilation or "stretch" receptors[4] that helps to control cough where cough begins—at points of irritation in the chest.

Controls cough at the cough center in the medulla

Spinal reflex arcs were studied for the inhibitory effect of TESSALON on the transfer of afferent cough impulses to the efferent branch of the cough reflex.[5] The administration of TESSALON inhibited reflex transmission, when the afferent nerve was stimulated electrically. With this "damping" effect on the cough center in the medulla, TESSALON controls cough centrally, as well as peripherally.

Thins sputum

TESSALON controls cough frequency without interfering with productivity or expectoration. In fact, sputum is usually thinner, easier to raise.[1]

Effect of TESSALON on sputum[1]

Amount		Consistency	
Less	32 patients	Heavier	3 patients
More	2* patients	Lighter	27 patients
Same	16 patients	Same	20 patients

*These patients noted more but lighter sputum.

Increases vital capacity

Respiration usually increases both in depth and volume during TESSALON treatment.[6] In one study,[7] patients with chronic respiratory disease, with and without bronchospasm, showed a mean increase of 19.7 per cent in vital capacity after a 2-week course of TESSALON.

Improves exercise tolerance

By inhibiting stretch receptor activity, and by increasing air intake, TESSALON enables patients to tolerate exercise or work better, eliminates many paroxysms of coughing.

Relieves dyspnea

Farber and Wilson[8] note that one of the important contributions of TESSALON to cough therapy is "...its action as a reliever of dyspnea in some patients." Shortness of breath, wheezing, weakness, "blackouts" are not likely to trouble the patient treated with TESSALON.

Fast, prolonged action

The cough suppressant effect of TESSALON starts rapidly—usually within 15 to 20 minutes. The duration of effect is prolonged—usually from 3 to 8 hours.

Indications

TESSALON is indicated in acute and chronic cough.

ACUTE: Common cold, Bronchitis, Pneumonia, Upper respiratory infection, Pleurisy, Spontaneous pneumothorax, Bronchial irritation provoked by gases and foreign bodies. CHRONIC: Pulmonary emphysema, Bronchitis (emphysematous, asthmatic), Bronchial asthma, Tuberculosis, other chronic pulmonary diseases, Pulmonary or mediastinal tumors

PROCEDURES: Bronchoscopy and bronchography, Thoracentesis, Thoracic surgery

Dosage

ADULTS: Average dosage is one Perle (100 mg.) t.i.d. If necessary, or where cough is refractory, up to 6 Perles (600 mg.) daily may be given.

CHILDREN UNDER 10: One Pediatric Perle (50 mg.) t.i.d. is the usual dosage.

The Perles should be swallowed without chewing, and, if necessary, with a liquid. Release of TESSALON from the Perle in the mouth produces a temporary local anesthesia of the oral mucosa.

Side Effects

TESSALON is well tolerated. Only occasionally have side effects been reported. Skin rash, nasal congestion and a vague "chilly" sensation have been mentioned. In rare instances, gastrointestinal upset, constipation or sedation have been observed. No adverse effects on respiration, kidney or liver function tests, blood count or urinalysis were reported.

Supplied

Perle form (liquid-filled gelatin spheres) provides speed of liquid medication—convenience and dosage accuracy of capsule medication. In two strengths: 100-mg. Perles (yellow), for adult use; 50-mg. Perles (red), for children under 10.

Samples available on request.

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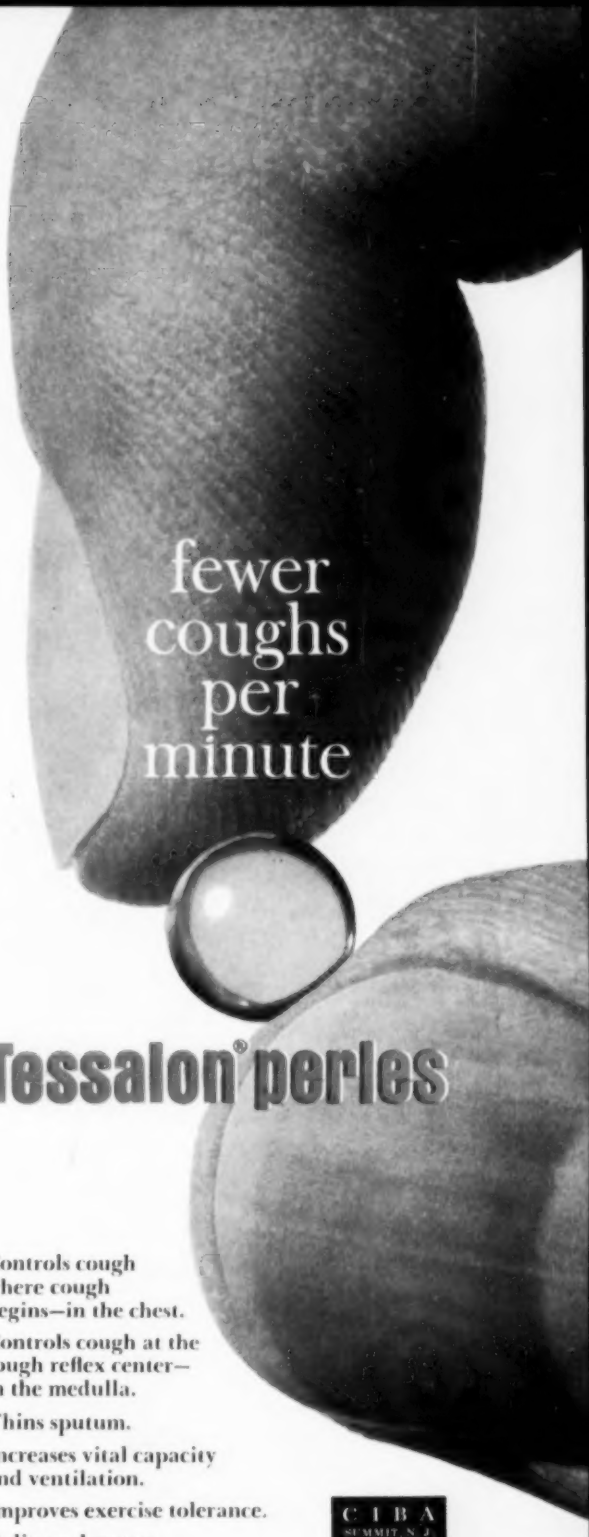

Tessalon® perles

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Controls cough at the cough reflex center—in the medulla.

Thins sputum.

Increases vital capacity and ventilation.

Improves exercise tolerance.

Relieves dyspnea.

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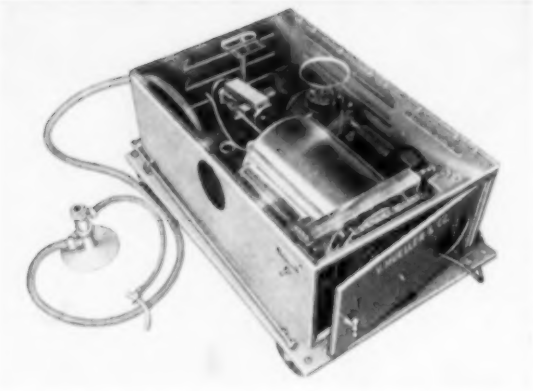

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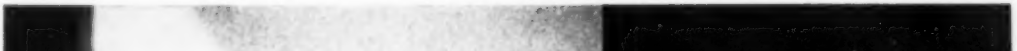

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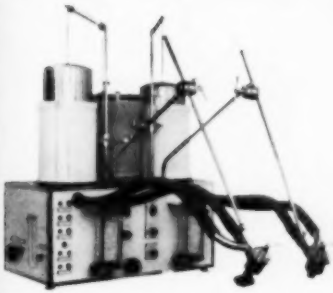

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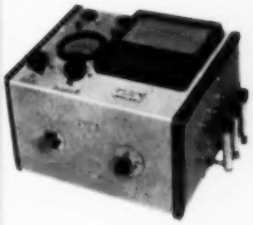

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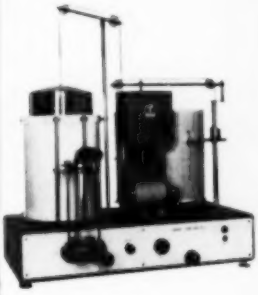

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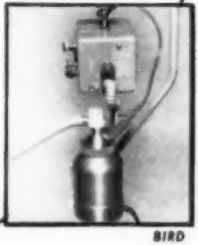

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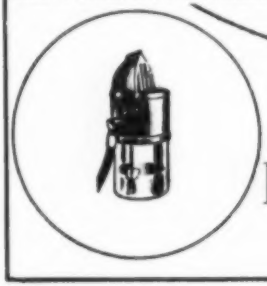

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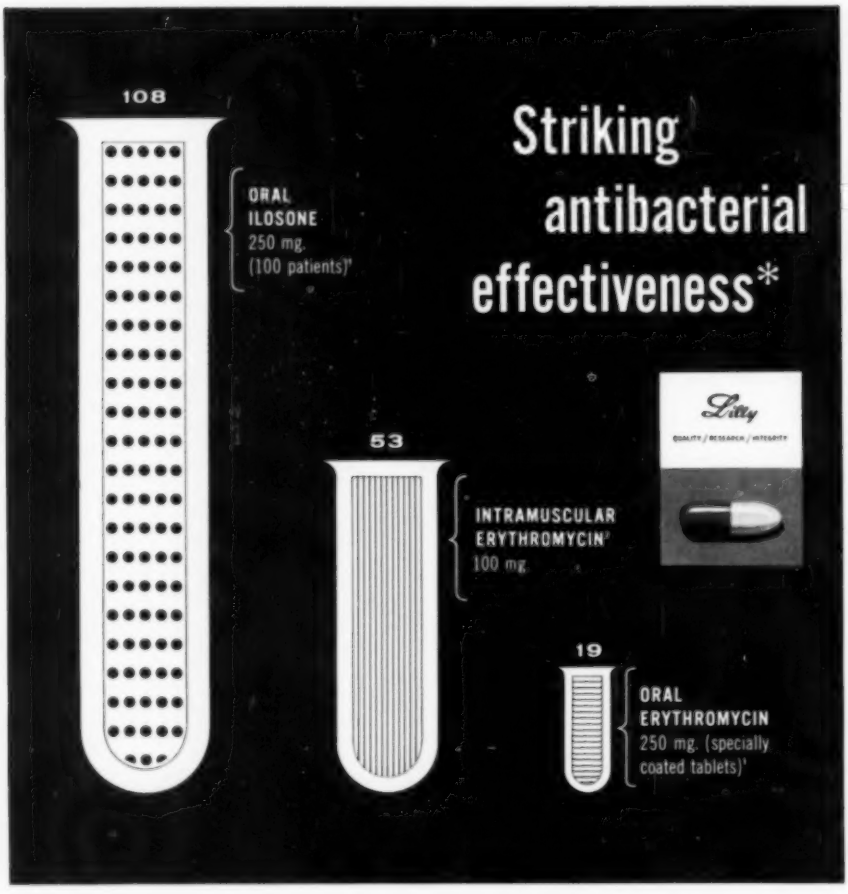

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DISEASES of the CHEST

VOLUME XXXV JANUARY, 1959 NUMBER 1

The Place of Pneumothorax in the Present-Day Management of Pulmonary Tuberculosis

GÖSTA BIRATH, M.D., F.C.C.P.*

Gothenburg, Sweden

In recent years, artificial pneumothorax has suffered a bad name and lost its popularity. One antagonist to its use has called the pneumothorax needle the most dangerous weapon ever placed in the hands of a physician. If the physician in question is indiscriminate or inexperienced, the phrase undoubtedly has a ring of truth about it. It is certainly not surprising that a swing away from the method has taken place when one recalls that it has been only a decade or so since it was considered a tool for the treatment of all tuberculosis—acute or chronic, exudative or productive. It was axiomatic that pneumothorax should be tried before thoracoplasty could be considered. Even though it was realized that the method met with more success in certain forms of the disease than in others, attempts to delineate indications were few. It wasn't at all unusual for half of a sanatorium's clientele to be under this form of treatment.

Now the use of pneumothorax has decreased even among its supporters. Essentially there are two reasons for this. One is the increasing use of resection. In Holland, for example, 44 per cent of active procedures against pulmonary tuberculosis consisted of pneumothorax management in 1949, whereas in 1955 the proportion was down to 3 per cent. Holland can be considered the country in Europe where recourse to resection has been taken more than anywhere else, and the use of pneumothorax has decreased in a corresponding degree. In Sweden the figures for the same years were 64 per cent (1949) and 21 per cent (1955).

The good results obtained from long-term, careful antimicrobial chemotherapy have also been associated with the decrease in the incidence of pneumothorax inductions and are another reason for the decline in this form of treatment.

Indications for artificial pneumothorax today

Is there any reason for the use of pneumothorax nowadays? The writer believes that such is the case until we have new weapons of proved superiority, so there will be no need of any other methods. He does not feel that we have yet reached thus far for the following reasons:

1. Although the relapse rate in pulmonary tuberculosis after adequate chemotherapy on the whole seems low we do not know how the prognosis is in conditions with greater relapse tendency such as alcoholism, diabetes, etc.

*Renströmska Sjukhuset.

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2. Not all cases heal on chemotherapy because of intolerance against the drugs and bacterial resistance.

It is inescapable that tuberculosis has been characterized by a large relapse risk. In Holland between the years 1953-1955 one third of all patients with pulmonary tuberculosis of Nederlandse Sanatorium-Verzekering had relapsed. In Sweden the percentage is probably even higher. In any case, of all patients admitted to Swedish sanatoria in 1955 not less than 69 per cent were readmissions.

In order to meet the problem of relapse radically most lung specialists agree that chronic, localized lesions often must be resected and few neglect a course of long term chemotherapy in such a case if that is possible to carry out. However, it is of great importance that the disease even in its acute phase be treated so that it heals and a chronic lesion be prevented.

Consequently, acute tuberculosis calls for radical treatment no matter how small its extent. To this end we have available:

1. Chemotherapy.
2. Bed rest.
3. Temporary collapse procedures.

Only in exceptional cases is resection indicated in this stage of the disease.

It is possible that most acute cases heal with chemotherapy alone. Certainly the minimal cases in particular often have no need of bedrest. However, we have found it wise to hospitalize them for a period of two or three months in order:

1. To test out the adequate chemotherapy.
2. To educate the patient in the disease and its treatment.
3. To prevent spreading of the disease during its contagious phase.

As everybody knows, the patient often does not tolerate the drug and suffers from gastric disorders, allergy and, last but not least, toxic reactions to isoniazid, i.e., signs of polyneuritis and—in this country often—mental disturbances.

When the ordinary methods of the above-mentioned reasons are likely to be unsuccessful we consider pneumothorax still to be useful:

1. When the disease process is due to drug-resistance bacilli.

2. When the patient demonstrates intolerance against one or more of the antimicrobials in a combined regimen, preventing a successful course of long term chemotherapy.

3. When one can anticipate that the drugs will not be taken with the necessary dosage and regularity. Certain patients with psychopathia, alcoholism and some youngsters fall into this group.

We have noticed in this connection that PAS has found its way into the toilet instead of the patient and this possibility has to be kept in mind when bacterial resistance arises unexpectedly against one of the other drugs in the patients combined regimen.

4. When the relapse risk is especially great, as in cases complicated with diabetes, alcoholism, peptic ulcer, after gastric resection, etc.

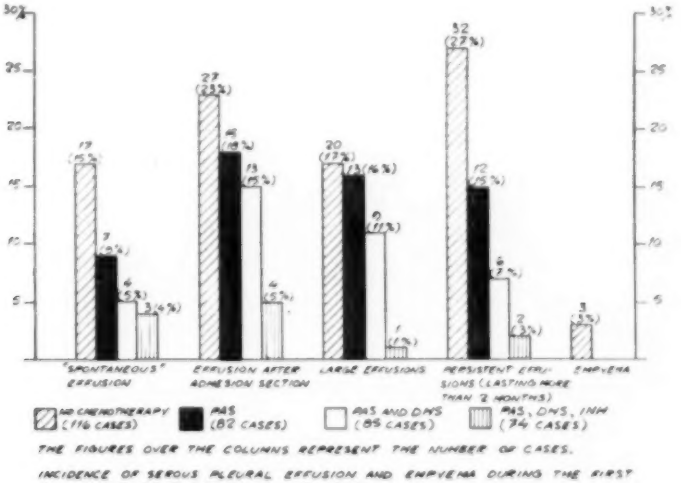
As pointed out above, the form of disease calling for temporary collapse is the fresh lesion. Classically this is the infiltrate with cavitation even though this indication is not so compulsory as it once was. In addition pneumothorax is of value in the multilocular forms of fresh tuberculosis.

In many centers antimicrobial drugs treatment has been used to the exclusion of pneumothorax so that the availability and technique of the latter has almost been forgotten especially when it comes to adhesion section and the management of exudate formation, etc. This exclusion seems to me premature.

On the basis of this indication, pneumothorax is now used in our hospital in about 4 - 5 per cent of all cases, which certainly is a small amount. However, it keeps the doctors familiar with the method and it provides a reliable treatment of such patients, who do not respond successfully to the ordinary methods.

Effectivity of pneumothorax treatment

Is pneumothorax management really effective? The disdain with which it has been regarded in recent years has been accompanied by a tendency to forget the indisputably good results which have been obtained with its use. Jennings, Mattill, and Nemec[1] reported in 1940 that clear improvement took place in their series of 1,000 cases if the diseased area was successfully collapsed. The other cases, including control groups with total pleural synechia, those with unsatisfactory collapse, and those with collapse of other areas than the diseased all had a poorer outcome; in the ideal group no less than 80 per cent were capable of returning to work and the mortality rate was only 4 per cent 1 - 20 years later. Sinding-Larsen[2] reported a group of 158 cases who were treated at least two years with an effective pneumothorax, there were no less than 89 per cent at work and free from bacilli at the time of a follow-up study two to 25 years after the conclusion of the treatment. Most of these patients were examined after more than 10 years. Only one relapse at the original site of disease occurred in these 158 cases. Törning and Springborg[3] followed up 107 cases who had had pneumothorax treatment for unilateral tuberculosis involving about ¼ of the lung. All of these cases had a free lung during the course of treatment; three to 13 years later 80 per cent of the cases were considered cured.



These results must be remembered moreover in the light of the fact that they were obtained in the days prior to chemotherapy. In addition patients were included who had chronic lesions which do not respond well to collapse treatment, since it was not customary at that time to exclude such patients from pneumothorax treatment.

In addition these results must also be considered against the background of the poor prognosis of the so called open tuberculosis at that time. In general most of those with cavitary disease were dead within five years. In the series reported by Berg[4] only 20-30 per cent were alive 10 years after open tuberculosis was diagnosed. Other statistics from that period are even worse.

It has been predicted that antimicrobial treatment and pneumothorax from a theoretical standpoint should be antagonistic, but convincing data in support of this are not at hand. Cavity closure moreover takes place in a different way as a result of chemotherapy as compared to collapse-induced healing. In the latter case, healing occurs from the draining bronchus which closes off the cavity, while healing under the influence of antimicrobials is not associated with this blocking action (Auerbach, Katz & Small[5]). What this means to the prognosis is not yet clear.

Prevention of Complications

Critics of the method maintain that pneumothorax is associated with serious complications. It is well known that exudate in the pre-chemotherapy era often formed and caused severe ipsilateral function loss. By certain authors these complications have been contrasted to the success of resection-chemotherapy. Such a comparison can hardly be correct. If one compares on the other hand pneumothorax cases from recent years in which antimicrobial drugs were also used, one finds that the incidence of complicating exudates has become insignificant. Pre-chemotherapy pneumothoraces were associated in 50-60 per cent of cases with the formation of an exudate within 6 months after induction; nowadays the corresponding figures are only 3-5 per cent.[6] In addition the exudate now encountered is almost never large or of long duration, which means that empyemas have completely disappeared and function-depressing peels seldom form. See graph on page 3.

In those cases where pleural reaction does occur in the present day form of pneumothorax management, it is possible to prevent crippling of the lung's function with special measures.[7] The main principles of this form of management consist of frequent thoracenteses and prompt expansion of the lower lobe, which is usually free of parenchymal changes and does not need to be kept collapsed. Sometimes streptomycin or isoniazid are given intrapleurally one to two weeks. At the same time the patient is instructed to lie on his good side and perform abdominal breathing exercises, usually a series of 15 deep breaths every hour, in order to keep the diaphragm mobile, reexpand the lower lobe and prevent adhesion of the diaphragm to the chest wall. By using small frequent refills during this period, the collapse of the diseased upper lobe can be maintained.

CONCLUSION AND SUMMARY

Pneumothorax treatment of pulmonary tuberculosis today has stricter indications than before, but if used with the proper technique, it provides

good results. The complications of exudate and empyema and function-reducing pleural peels are now minimal thanks to concomitant chemotherapy and the technique of including selective collapse while maintaining good diaphragmatic mobility. The coming few years will show whether or not the inclusion of pneumothorax as part of an active program for the prevention of relapse is justified.

RESUMEN

El tratamiento con neumotórax en la tuberculosis pulmonar, hoy tiene indicaciones más estrictas que antes, pero si se usa con técnica adecuada proporciona buenos resultados. Las complicaciones consistentes en exudados y empiema y reducción funcional por engrosamiento pleural son ahora mínimos gracias a la quimioterapia concomitante y a la técnica de incluir un colapso colectivo conservando la movilidad diafragmática. Los pocos años próximos mostrarán si la inclusión del neumotórax está justificada o no como parte de un plan activo para prevenir las recaídas.

RESUME

Le traitement de la tuberculose pulmonaire par le pneumothorax a aujourd'hui des indications plus réduites qu'auparavant, mais s'il est utilisé avec une technique convenable, il offre de bons résultats. L'épanchement liquidien, la purulence, la diminution de la liberté pleurale réduisant la fonction sont maintenant des complications rares. On les évite grâce à l'emploi simultané de la chimiothérapie et du procédé consistant à utiliser un collapsus électif qui cependant conservent une bonne mobilité diaphragmatique. Les quelques années à venir montreront si l'association du pneumothorax au traitement dans le but de prévenir les rechutes est justifié ou non.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNG

Die Behandlung der Lungentuberkulose mit Pneumothorax besitzt heute striktere Indikationen als früher; wird sie aber mit entsprechender Technik gebraucht, so liefert sie gute Ergebnisse. Di Komplikationen an Exsudat und Empyem und funktionshindernden Pleuraschwarten sind heute minimal, dank der gleichzeitigen Chemotherapie und der Technik des selektiven Kollapes unter Aufrechterhaltung einer guten Zwerchfellverschieblichkeit. Die kommeden Jahre schon werden zeigen, in wie weit die Einbezeihung des Pneumothorax als Teil eines aktiven Programmes zur Verhütung von Rückfällen gerechtgertigt ist.

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Aspiration Pneumonia*

IRVIN R. BESMAN, M.D.** and HAROLD A. LYONS, M.D., F.C.C.P.†
Brooklyn, New York

Aspiration pneumonia is a pulmonary problem that sometimes presents diagnostic difficulty. These types of pneumonia are frequently neglected in the differential diagnosis of pulmonary lesions because they are not considered. There are instances in which these conditions have been diagnosed as neoplasm of the lung and only on subsequent resection or pneumonectomy was the true nature of the condition established.[14,18,19,30] Attention to this group of pulmonary diseases is important because if the diagnosis is correctly established, needless empirical forms of prolonged therapeutic measures can be eliminated; treatment may be recommended which may prevent the development of (1) chronic suppurative disease, (2) the further progression of the pulmonary involvement, by removal of the etiologic agent, and (3) the serious development of far-advanced irreversible pulmonary emphysema, fibrosis, and finally (4) cor pulmonale.

This report includes several etiologic types of aspiration pneumonias and the problems they propose. We have classified the aspiration pneumonias on the basis of etiology into (1) lipoid pneumonia—aspiration of oils, e.g. mineral oil, etc., (2) aspiration of food and gastric secretions, (3) aspiration of foreign bodies, e.g. teeth, etc. and (4) aspiration of infected material from the mouth and throat.

Lipoid Pneumonia

Guieyse-Pellissier[1] in 1920 observed the results of intra-tracheal instillations of various oils in the lungs of dogs and rabbits and is credited with the first reports of lipoid pneumonia in the literature. In 1925 Laughlen[2] reported the first clinical case of lipoid pneumonia in children who had received naso pharyngeal instillations of menthol and argyrol in an oil base and died of cystic pneumonia. Pinkerton[3] in 1927 reported several cases of lipoid pneumonia in infants and found six cases in 200 consecutive autopsies. Since these earlier reports there have been many collective series of clinical and autopsied cases of lipoid pneumonia.[4-9] The incidence of lipoid pneumonia found by these investigators varied from 0.3 to 14.6 per cent.

Pinkerton[3] demonstrated that various oils cause different reactions in the lung. He showed that animal oils (cod liver oil, milk fat) produced fibrosis and giant cell formation together with necrosis and edema depending on the quantity of free fatty acid liberated by hydrolysis. Vegetable oils (sesame, olive, poppyseed) produce practically no reaction. Mineral

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oil (liquid petrolatum) causes a marked macrophagic response together with giant cell formation and eventually fibrosis.

Experimental studies have shown that mineral oil does not elicit a cough reflex and mechanically hinders ciliary action.[2,4,10]

Moel and Taylor[11] stated that oil entered the lung in three ways: (1) through the mouth, (2) instilled into the nose as drops, and (3) through tracheotomy as a lubricant. Pathologic processes which interfere with the normal cough, palatal or swallowing reflexes are important predisposing factors in the aspiration of mineral oil. These have been listed as (1) neurologic conditions associated with dysphagia, parkinsonism, pseudobulbar palsy, etc., (2) neoplastic or other processes involving the mouth and throat such as cleft palate, (3) debilitating conditions, whatever their cause, and (4) cardiospasm.[22, 40] Volk and his associates,[7] found the highest incidence of lipoid pneumonia in their study of 389 chronically ill patients. A common practice in hospitals is to give debilitated, bedridden patients mineral oil.

Schneider[12] demonstrated the pulmonary hazards of habitual use of mineral oil in the apparently healthy ambulatory individual. Oily nose drops, long known to be a cause of lipoid pneumonia since 1927 when Laughlen[2] reported his cases, are still being used and were the cause of lipoid pneumonia in two of our cases.

The deposition of oil into the lungs depends almost exclusively on mechanical factors.[13] Thus there is a tendency, as with all types of aspiration pneumonias, for localization in the most dependent portions of the lungs, the posterior basal divisions of the lower lobes. Bilateral is more common than unilateral involvement. Involvement of the upper lobes is assumed to be due to the insufflation of mineral oil in the supine position or prior to retiring.

Mineral oil, upon reaching the alveolar spaces, sets up a tissue reaction of the foreign-body type which may progress with fibrosis and vary in size from small nodules to large masses (paraffinoma).

A great many authors[7, 17-19, 27] have commented on how closely the lesion resembles carcinoma grossly. Berg and Burford[14] reported a case where frozen section gave positive diagnosis of carcinoma. The question whether lipoid pneumonia is actually carcinogenic has been raised by Shimkin[15] who produced pulmonary carcinoma in mice by intratracheal instillations of hydrocarbons. Twort and Lyth[16] found that several fractions of mineral oil produced primary lung tumors in mice.

Several authors[20, 21] reported on methods by which mineral oil can be demonstrated in tissue.

In the majority of patients there are no signs or symptoms, even with extensive pulmonary involvement. Most instances of the disease are usually diagnosed at autopsy. A routine chest x-ray film may bring the patient into the hospital for a diagnostic workup. Symptoms were present in half of the cases reported by Freiman and his associates.[5]

The usual symptom is cough which is chronic and occasionally productive. Chest pain is uncommon as is hemoptysis, but this has been reported. It is uncommon for complications of bronchiectasis or suppuration to occur.[12] Bronchopneumonia, however, is not uncommon and repeated episodes

have been described in the literature.[12] As pulmonary fibrosis becomes more extensive due to repeated intake of liquid petrolatum, the patient develops dyspnea and hacking cough. Clubbing is said to be uncommon in uncomplicated lipoid pneumonia. Cor pulmonale is a rare complication and is due to obliteration of the vascular bed by the granulomatous lesions. This has been reported on by Steinberg and Finby.[22]

Roentgenographically, two main types of lipoid pneumonia can be distinguished: (1) diffuse type and (2) circumscribed, or nodular type. These have been excellently described in detail by Volk, Losner, Lewitan and Nathanson[23] and Hampton and Bickham[24] who reviewed the x-ray film findings in 35 cases. The diffuse type is more common (70 per cent) and is characterized by diffuse, fine linear spun glass densities, the periphery of which is faded.[24] Several lobes are usually involved and roentgenographically this cannot be differentiated from any other pneumonia. Late in the disease, fibrosis occurs and with areas of contraction and compensatory emphysema, a "ground-glass" appearance may be seen.

The nodular type is characterized on x-ray film by a homogeneous soft tissue density varying from several centimeters to large masses (paraffinoma).[23,24] The peripheral limits may be sharply demarcated but occasionally are irregular with fine projections into adjacent tissues. A valuable roentgen sign described by Hampton, Bickham and Winship[24] is the contrasting feathering of density at the hilar aspect compared to the peripheral limits which is due to lipophages along the peripheral lymphatics. Pleural effusions are rarely encountered.

Lipoid pneumonia must be considered in all basal pneumonic processes of undetermined etiology. Suspicion should be great in bilateral lower lobe processes found on routine roentgenograms of adults with few complaints. The diagnosis should also be entertained in all bronchopneumonias which do not resolve.

The chief problem arises where the lesion is unilateral and must be differentiated from neoplasm. The diagnosis must be made by clinical, radiologic, and cytologic approach. A history of habitual ingestion of mineral oil in patients with poor or absent gag reflexes, or who use oily nose drops, should make the clinician suspicious. Lesions which are located in the recognized aspiration sites: basal segment of the lower lobes, right middle lobe would be grounds for consideration of this diagnosis.

Losner, Volk et al.[25] proposed a cytologic study on sputum. The patient is placed on a fat-free diet beginning four days prior to collection of sputum as well as throughout the collection. This is important because fat in the diet can lead to false positive results.[26] Twenty-four hour collection of sputum should be done daily for three to five consecutive days. A drop of sputum is placed on four glass slides and a drop of saline is added to make a thin spread. Slides are dried in the incubator. Two slides are stained with Sudan IV and two with Wright's stain. The sputum is positive when the Wright's stain shows characteristic macrophages containing clusters of vacuoles and also if vacuoles stain orange-brown with the Sudan IV. (Fig. 1)

Lung aspiration biopsy has been performed by many workers[27,28] using a 16-17 spinal needle under fluoroscopy and has proved valuable in experienced hands, but we do not recommend this diagnostic technic.

There is no real therapy for lipoid pneumonia except a prophylactic one and discontinuance of use of mineral oil. Oily lipoid pneumonia will resolve if the source of infiltration is stopped in time.[5] If secondary pulmonary suppurative bronchiectasis or recurrent attacks of bronchopneumonia occur, surgery should be considered. The question of possible carcinogenic properties of mineral oil might also lead to a consideration of early surgery in lipoid granulomas.[29]

Case 1: W. A. A 41 year old colored man was admitted with a history of being well until two to three months prior to admission when he developed weakness on exertion, dyspnea, night sweats, weight loss of 25 lbs. and cough productive of several cupfuls of thick yellowish sputum daily. He developed sharp pain in the left chest aggravated by coughing and deep inspiration. He had a "normal" chest x-ray film several months previously. On subsequent questioning he gave a long-standing history of using oily nose drops.

Physical examination revealed a temperature of 103° F., and blood pressure of 115/75. Chest findings revealed diminished tactile fremitus, dullness to percussion, decreased breath sounds over the left lower lobe. Clubbing of the fingers was present.

The roentgenogram of the chest revealed a hilar mass with infiltration in both lower lung fields (Fig. 2).

The white blood cell count was 10,200 per cumm. with 81 per cent polymorphoneutrophils, 17 per cent lymphocytes and two monocytes. Hemoglobin was 7.5 gms. Urinalysis showed many white blood cells on microscopic examination. Tests with first and second strength purified protein derivative were negative. Sputum culture revealed normal respiratory flora.

He was started on penicillin and gantricin®, for urinary tract infection. He continued to have low-grade fever. On bronchoscopic examination the left bronchus was found to be filled with thick yellowish secretions. The sputum obtained with Sudan IV revealed many macrophages containing lipoid bodies (Fig. 1).

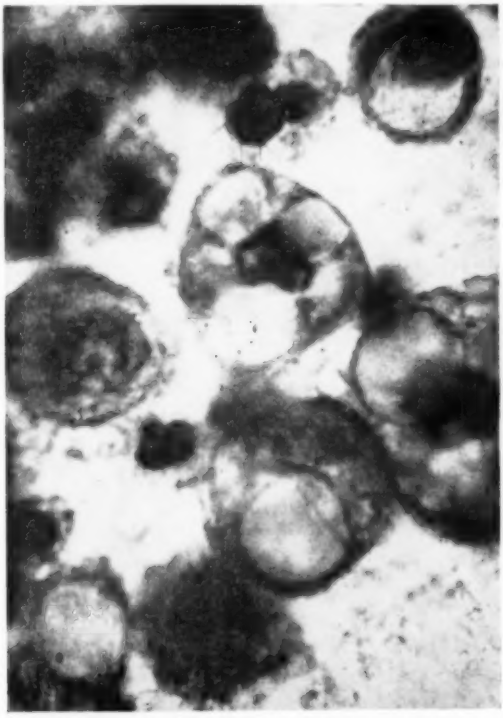

FIGURE 1: Slide stained with Papanicolaou and Sudan stain shows characteristic macrophages containing clusters of vacuoles indicative of positive sputa.

The roentgenogram of the chest on discharge was completely clear (Fig. 3).

Case 2: J. B. A 56 year old white man was found to have a carcinoma of the larynx. He was operated upon and tracheotomy had to be performed. The chest film at this time was completely normal except for the tracheotomy (Fig. 4). Nine months later he entered the hospital with dyspnea and cough productive of one cupful of yellowish sputum daily.

Physical examination of the chest revealed widespread fine rales all over the thorax. Pulmonary edema due to cardiac failure was present and was treated by digitalization. The pulmonary edema cleared, but the chest film (Fig. 5) continued to show bilateral lower lobe infiltration greater on the left. The sputum on bacteriologic examination had a mixed respiratory flora. An intensive search for the etiology of his pulmonary disease was not fruitful. During ward rounds one day, the etiology was established when he was found to be lubricating his tracheotomy tube with vaseline. This was confirmed by the appropriate sputum cytological study.

Food and Gastric Aspiration Pneumonia

Ernst Becker[31] in 1887 was one of the first to cite aspiration of the stomach contents as a cause of bronchopneumonia. He described this in relation to postoperative complications and to gastric disease. Other authors[32-35] stressed the importance of postoperative aspiration of stomach contents.

The pathologic changes that occur after aspiration of gastric contents were observed experimentally by Moran[36] who injected intratracheally in 60 guinea pigs a variety of common cooked foods. He found that a generalized pneumonic reaction occurred in addition to a severe local reaction immediately around the food. This developed within two to six hours and consisted of polymorphonuclear and red blood cells. Numerous large granulomatous areas were noted at the end of one week. Fibrosis was seen at the end of two months.

Teabeaut[37] found that the degree of tissue response seems to parallel the pH and volume of injected hydrochloric acid solution between pH 2.4 and 1.5

In a study of experimental aspiration pneumonia in rabbits, Moran[38] found it resembled the clinical form in people. He first observed a neutro-

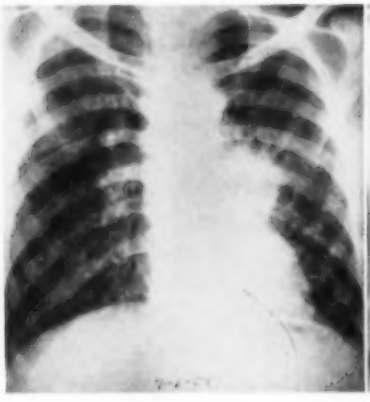

FIGURE 2

FIGURE 3

Figure 2 (Case 1): The posterior-anterior chest film taken on admission. There is a large dense shadow in the left hilar area and diffuse areas of infiltration in both lower lung fields.—*Figure 3* (Case 1): This film shows completely normal lung fields three months later after cessation of oily nose drops.

phillic reaction followed later by a granulomatous lesion with a giant cell reaction and fibrosis.

Smith and Moran[39] studied the effect of carbohydrate solutions and found an aspiration pneumonia similar to but as severe as food aspiration pneumonia.

Clinically, aspiration of gastric contents may occur in any patient who is unconscious or in coma with a depressed gag reflex. The earlier workers[31,34] cited this in postoperative patients who were deeply anesthetized and who then vomited. Intoxicated persons, those with head trauma, and metabolic coma as diabetes, are likely candidates for aspiration pneumonia.

Some lesions of the esophagus predispose to this type of aspiration pneumonia. Anderson and his associates[40] in 1953 studied the records of 601 patients with cardiospasm at the Mayo Clinic during the 12 year period, 1935-1946 and found 63 (10.5 per cent) had pulmonary complications. The most common lesion was aspiration pneumonia. Esophageal diverticula, stricture, and neoplastic lesions have also been incriminated.

Clinically, there are two phases of this type of aspiration pneumonia:[37] (1) the acute reaction, in which bronchial obstruction and generalized pulmonary edema occur and may cause sudden death, and (2) the chronic reaction of chronic bronchial obstruction and pulmonary fibrosis.

When gastric contents are aspirated, obstruction of the airway may occur, depending on the size of the particle. Pulmonary edema occurs early due to the irritant effect of the material.[34, 36] Bronchoscopy is indicated immediately in order to aspirate the particulate matter and fluid from the respiratory tract. The pathogenesis of aspiration pneumonia is not dependent on bacteria; therefore, antibiotics are not of primary therapeutic value. These drugs are used to prevent secondary infection.

There is little to be done for the chronic type of reaction where granulomatous lesions and fibrosis have already occurred. Lesions and disorders of the esophagus should be corrected.

FIGURE 4

FIGURE 5

Figure 4 (Case 2): Posterior-anterior film of the chest taken after total laryngectomy and tracheotomy. This film reveals the tracheotomy tube and is otherwise a normal chest film.—*Figure 5* (Case 2): Posterior-anterior film of the chest taken nine months after the previous chest film reveals tracheotomy tube present. There is extensive infiltration in the left lower lung field and some infiltration in the right lower lung field.

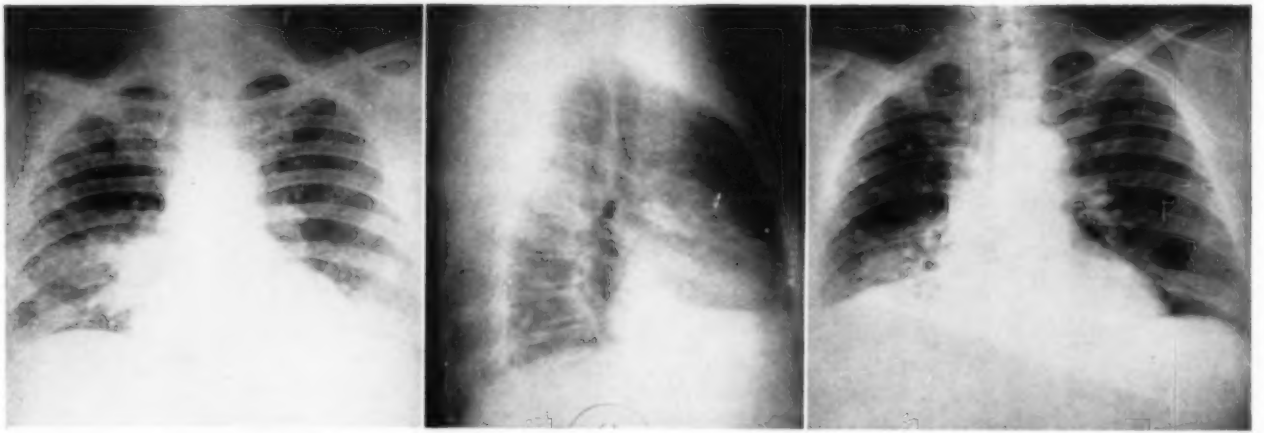

FIGURE 6 FIGURE 7 FIGURE 8

Figure 6 (Case 3): Posterior-anterior film of the chest taken on admission. Productive foci of infiltrations are present in the right upper lobe segments. An area of dense infiltration is present in the lateral segment of the right middle lobe. Some infiltrations also are present in the superior segment of the right lower lobe. Bronchogenic spread to the basilar segments of the right lower lobes is noted. There is no contralateral parenchymal spread.—*Figure 7* (Case 3): Right lateral roentgenogram of the chest taken on admission revealed an increase in density involving the anterior and medial basal segments of the right lower lobe suggesting possibility of an atelectasis secondary to a possible bronchogenic block.—*Figure 8* (Case 3): Posterior-anterior film of the chest taken three months later after second admission revealed an increased density at the right base with some elevation of the diaphragm suggesting the possibility of an intra-pulmonary fluid collection. There is an infiltration in the basal segments of the right lower lobe.

Case 3: P. B. A 46 year old colored man was admitted to the Kings County Hospital for the first time because of hemoptysis of four days' duration. He gave a history of cough productive of two ounces of yellowish mucoid sputum daily for the past year. He also had anorexia and a 20 pound weight loss over several months. He smoked two packs of cigarettes daily.

Physical examination revealed that his blood pressure was 130/80 and his temperature was 98.6° F. Chest findings revealed diminished tactile fremitus, dullness to percussion, decreased breath sounds and wheezing over the right lower lobe. No clubbing was noted. The chest film on admission (Figs. 6, 7) revealed an area of dense infiltration in the lateral segment of the right middle lobe. Some infiltration also was noted in the superior segment of the right lower lobe. There was a bronchogenic spread to the basilar segments of the right lower lobe. The productive focus of infiltration was at the right upper lobe.

Papanicolaou smears of sputum were Class II. Bronchoscopy was performed and was not considered ideal due to his excessive coughing. A mulberry-shaped tumor was seen by the bronchoscopist proximal to the level of the right middle lobe orifice which bled easily. Biopsy was taken and reported as chronic inflammation. Papanicolaou smears of bronchial secretions were Class II. The impression was bronchogenic carcinoma. He, however, refused surgery and left the hospital against advice.

He was readmitted three months later with a history of the cough becoming worse. Dyspnea and hemoptysis were also present. The physical examination of the chest again revealed a lag on respiration on the right, diminished breath sounds in the right lower lung field. The chest film at this time showed an infiltration in the right lower lobe (Fig. 8). Bronchoscopy was repeated and was considered unsatisfactory due to excessive coughing. The right lower lobe bronchus was occluded with a nodular mass which bled easily. The biopsy report was chronic inflammation.

Right pneumonectomy was performed. The right lower lobe was found to be completely atelectatic and consolidated. The right middle lobe was atelectatic with bronchiectasis. The right upper lobe showed a marked degree of saccular bronchiectasis. Two pieces of chicken bone were found. One lodged in the posterior basal segment of the right lower lobe and the other in the lateral segment of the right middle lobe.

Aspiration of Foreign Bodies

Aspiration of foreign bodies is more common in children. The most usual foreign body aspirated is some form of nut.[41,42] In adults, the aspiration of dental materials appears to be most common.[42]

The pathologic changes in the lung depend on the site of lodgement, length of time that elapses before its removal, and the chemical and physical characteristics of the substance. Some substances cause little or no reaction. Smooth metals which are not obstructing produce little or no reaction.[43] Rough metals producing obstruction and corrosion cause local inflammatory reaction and mucosal swelling. All vegetal substances irritate the mucosa causing a diffuse reaction, known as vegetal bronchitis[44,45] which occurs early with fever, toxemia and cough. When complete occlusion occurs, atelectasis results, and secondary infection takes place producing the "drowned lung" of Jackson.[45] That is, the natural passages are filled with pus. Abscess formation does not develop for many weeks or longer. However, Spain and Lester[46] have shown that a foreign body obstruction may produce irreversible bronchiectasis within three days. Lobar pneumonia is a rare sequella.[43]

Symptoms of foreign body in the lower respiratory tract are coughing, wheezing and dyspnea in varying degrees, regardless of the location.[47] However, the symptomless period that follows non-obstructive foreign bodies may confuse the observer. This period may range from a few days to months until signs of obstruction occur. This usually occurs particularly with smooth metallic foreign bodies. Jackson and Jackson had over 300 cases, in which, after initial symptoms, the patients were symptomless.[43]

In the larynx there is usually hoarseness, croupy cough respiratory obstruction may occur depending on the degree of laryngeal edema produced

or block by the foreign body. An audible whistle or stridor[48] may be heard. Direct laryngoscopy establishes the diagnosis.

In the trachea, there is an asthmatic wheeze,[49] an audible slap[44] as the foreign body in the trachea strikes the under surface of the vocal cords. This is best heard with the mouth open and the patient coughing. Stridor and retraction of the thorax[47] may be present. The chest film is usually negative. Kjellberg[50] noted in children where the tracheal rings are soft and compressible, that on inspiration in roentgenograms, the trachea below the foreign body becomes narrower because of the negative pressure, whereas the trachea above the foreign body becomes wider. On expiration, the lower trachea becomes normal or widened and above the foreign body the trachea is either normal or widened.

Foreign bodies most commonly lodge in the right main stem bronchus. The incidence over the left varies in the literature from 2:1 to 5:1.[41,44] The relatively straighter course, wider lumen, lesser angle of deviation from the tracheal axis and the situation of the carina to the left of the midline of the trachea is the cause.[44] Jackson and Jackson[41] state that right middle lobe foreign bodies are rare findings, only 3 per cent in their extensive series. Lodgement of the foreign body is usually temporary and it works its way peripherally.[45]

Cough, hemoptysis, metallic taste or odor may occur immediately or after a symptomless period. Foreign bodies of vegetable origin usually cause violent laryngotracheobronchitis with toxemia, cough and fever.[43,45] Organic foreign bodies such as bones and teeth produce symptoms of chronic pulmonary disease with clubbing of digits after many months or years.[52]

There are four classical types of valve-like obstructions of the major airways.[42,44,51,53,54] These are: (1) by-pass, where obstruction is partial and air is permitted to pass beyond the obstruction in either direction, (2) check-valve inspiration is unimpeded but expiration of air is prevented with resulting obstructive emphysema, (3) ball valve type produced by a movable obstructed object that may allow air at times to leave but not enter the lung with resulting atelectasis, and (4) stop valve type, where there is complete obstruction to the entrance of air with absorption of all air from the peripheral lung and subsequent atelectasis.

One process may be present early and pass into another when edema and fibrosis result, changing the pathologic physiology.

The physical findings thus depend on the type of obstruction present.[51,53,54]

Manges[55] roentgen ray studies on foreign bodies are classical. A radiopaque object may be visualized revealing its location and size. Fluoroscopy is valuable for demonstrating the type of obstructions.

Bronchography may be required to demonstrate a radiolucent foreign body.

Jackson and Jackson[41] state that only 2 to 4 per cent of foreign bodies are coughed up spontaneously, and these are usually cone-shaped objects like teeth. Bronchoscopy is indicated for removal of foreign bodies and this should be done as early as the development of edema may make removal difficult.

Case 4: M. O'S. A 40 year old white woman was admitted to Kings County Hospital with a history of fever, cough and chills. She was well until three days before admission when her symptoms suddenly began.

Her temperature was 103° F. She had dullness to percussion, and fine rales at the right lower chest.

The chest film (Figs. 9 and 10) showed a metalic density in the right lower lobe bronchus, with lower lobe pneumonitis.

The white blood count was 13,500 per cumm. with a differential count of 81 per cent polymorphoneutrophils.

The foreign body removed by bronchoscopy was a gold cap of a tooth. She received penicillin and streptomycin for five days.

In further questioning after the chest roentgenogram was reviewed, it was found that before her illness she had partaken of considerable beer. She was quite unaware of her activities on the particular night, but recollected that the next day the gold cap of one of her teeth was missing. This type of information should be intensively searched for when aspiration may be a possibility.

Inhalation of Infected Material

Fetid lung abscess is caused by the inhalation of infected material from the teeth or tonsils into the bronchial tree, the site of the ensuing abscess corresponding to the segment of the lung which happens to be most dependent at that time.[56] What is not greatly appreciated, however, is that the aspiration of infected material from the mouth and throat may not always produce a lung abscess but may result in a less severe pneumonitis. This latter manifestation is usually misdiagnosed as viral pneumonia or bronchopneumonia.

In 1939, Ramsay and Scadding[57] called attention to the transient opacities found on chest films in patients with mild upper respiratory tract infections. They felt that these opacities were due to segmental or lobar collapse of the lung resulting from aspiration of infected mucus and obstruction

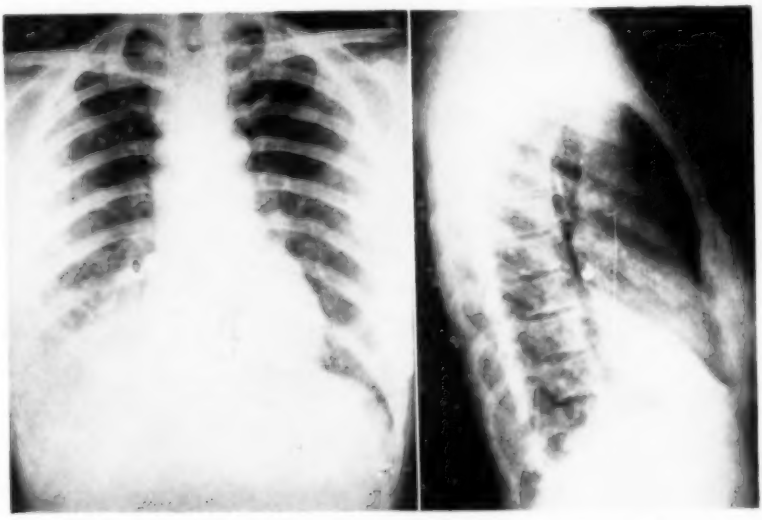

FIGURE 9 FIGURE 10

Figure 9 (Case 4): Posterior-anterior film of the chest on admission reveals metalic density in the right lower hilar region with secondary pneumonia.—*Figure 10* (Case 4): Right lateral chest film taken on admission reveals a metalic density located at the right lower lobe bronchus with secondary pulmonic infiltration in the anterior and medial basal segments.

of a segmental bronchus. They termed this benign aspiration pneumonia, since recovery invariably occurred.

Nicholson[58] in 1950 put forth his view that there is a whole range of aspiration pneumonias of varying severity in which suppuration occurs. He found that in his series at the Brompton Hospital predisposing factors were dental sepsis, extraction of teeth, anesthesia, and chronic bronchitis with aspiration of sputum. This type of suppurative aspiration pneumonia is most commonly seen by us in patients with poor oral hygiene, and states of deep unconsciousness where the gag reflex becomes depressed, produced by trauma, barbiturate intoxication, alcohol, or post-epileptic states.

Robertson and Morle[59] in 1951, reported on 500 cases of what they believed to be aspiration pneumonia following upper respiratory tract infections, which occurred in individuals undergoing military training. They showed that the clinical features of their patients were similar in all respects to those which have been frequently described in outbreaks of "primary atypical pneumonia." They cite that many cases of so-called "atypical pneumonia" prove to be aspiration pneumonia.[59,60]

On the chest x-ray film the most constant factor is localization to a specific lung segment. Localization, of course, depends upon which portion of the lung is most dependent at the time of aspiration.[56] When an individual lies on his side, inhaled material has an easy access to the posterior portions of the upper lobe. In the supine position the superior segment of the lower lobe is most readily affected. In the upright position the basal segment is most commonly involved.

Aspiration pneumonia, occurring in persons with poor dental hygiene, with poor or absent gag reflexes which do not go on to form solitary lung abscess, are usually diagnosed as lobar pneumonia or bronchopneumonia. They are usually acutely ill with high temperatures initially and then run low-grade fever for several weeks. Sputum is scanty at the onset but may

FIGURE 11

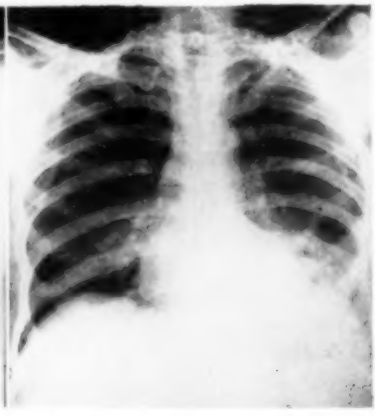

FIGURE 12

Figure 11 (Case 5) : Posterior-anterior film of the chest on admission reveals a homogeneous density in the left lower lung field. An area of infiltration is noted in the right lower lung field.—*Figure 12* (Case 5) : Posterior-anterior film of the chest taken one week after admission reveals a resolving pneumonia involving the basilar segments of both lower lobes. It is more marked on the left.

become copious later. Blood streaking is common. Chest pain is usually present and pleuritic in nature. Dyspnea was common in our patients. Sputum culture usually shows a mixed type of infection. X-ray films show slow clearing. Bronchiectasis is a frequent complication due to destruction of the bronchial musculo-elastic layer.

Bronchoscopy is indicated to rule out any obstructive bronchial lesions, for obtaining cultures and aspirating for therapeutic purposes. Penicillin and streptomycin, being bacteriocidal, are the agents of choice in the initial phase and the broad spectrum antibiotics only when there is clearing and symptomatic relief. Experience has demonstrated that antibiotics should be continued until complete clearing or completely stable roentgenographic appearances are obtained, or otherwise relapse becomes prominent.

Case 5: J. S. A 41 year old colored chronic alcoholic who had been drinking heavily for the past five years, was admitted to the hospital with a history of feverishness and a cough productive of scant amounts of yellowish sputum for two days. He had been drinking very heavily for the past week and drank himself into unconsciouness.

On physical examination his temperature was 104° F., blood pressure 134/100. He was acutely ill. His teeth were in poor condition and the gag reflex was definitely diminished. Dullness to percussion, diminished tactile fremitus and fine rales were noted over the left lower lung field. The white blood count was 13,100 per cumm. (82 per cent polymorphoneutrophils, 10 per cent lymphocytes and 20 per cent monocytes). On sputum culture. Neisseria, pneumococci, and streptococci were grown. The chest film taken on admission (Fig. 11) showed a density in the left lower lung field and infiltration in the right lower lung field.

He was placed on aqueous penicillin 30,000,000 units daily and his temperature fell to 101° F. Tetracycline (2 grams daily) was started 10 days after admission and penicillin was discontinued.

His chest x-ray films (Figs. 12, 13) showed slow resolution. He became afebrile 18 days after admission. Bronchoscopy was negative. The last chest x-ray film taken three months after admission (Fig. 14) was normal.

Discussion

The aspiration pneumonias are frequently unrecognized because they are not thought of by the physician. It is essential that the patient's history

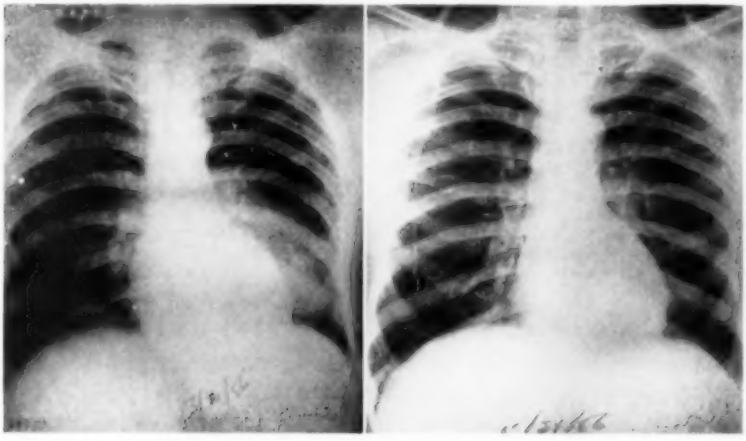

FIGURE 13 FIGURE 14

Figure 13 (Case 5): Posterior-anterior film taken one month after admission reveals resolution of the pneumonic process at the right lower lung field and left lung base.—*Figure 14* (Case 5): Posterior-anterior film of the chest taken three months after admission reveals a normal chest film.

be taken carefully to elicit any factors which may be indicative of pneumonitis, following the ingestion and/or aspiration of oily materials. In persons with a disorder of swallowing or laryngeal disorders, and in alcoholics, the possibility of aspiration pneumonia should be considered. When the cause of the pneumonic episode is established, then intelligent treatment may be carried out. This is especially true in those conditions which may produce serious and irreversible pulmonary damage.

There is also an immediate need for bronchoscopic examination of the patient, and for the search and removal of foreign material and bodies. A troublesome clinical course can often be explained when data such as this is available.

Again, mistakes may be made when the large dense granulomas of lipoid origin are thought to be bronchogenic carcinoma. But, here again, history, roentgenographic studies and cytologic studies will often prevent needless thoracotomy. It occasionally is necessary in some granulomas which persist to perform a resection because of persistent suppuration and disabling symptoms. Also, arguments may arise relative to the possible carcinogenic properties of the mineral oil in resolving lipoid granulomas.

When the roentgenologic examination of the chest shows evidence of pneumonitis in the commonly recognized sites for aspirated material, namely the most dependent portion of the lung at the time of the aspiration, this form of pneumonitis should be considered. Especially is this true when the clinical course is protracted or not easily explainable by the bacteriologic examination of the sputum. This is especially true when the patient is a chronic alcoholic or comatose and has normal respiratory flora on sputum examination. A poor response to the usual antibiotic therapy is characteristic. Poor dental hygiene, foul mouth, almost exclusively in these patients indicates an aspiration pneumonia due to infected material.

Interest has recently been displaced in right upper lobe unresolved pneumonias resembling bronchogenic carcinomas or pulmonary tuberculosis.[61] If one scrutinizes the reports, it is seen that this problem presents itself in alcoholics and probably represents instances of aspiration pneumonia.

SUMMARY

1. Aspiration pneumonia is a pulmonary problem that may present a great deal of diagnostic difficulty and is frequently neglected in the differential diagnosis of pulmonary lesions.

2. Aspiration pneumonia may result from (a) aspiration of oils-lipoid, (b) food and gastric secretions, (c) foreign bodies, and (d) infected materials.

3. Illustrated cases of each type are presented.

4. Aspiration pneumonias have a tendency for localization in the most dependent portions of the lungs at the time of aspiration.

5. Lipoid pneumonia should be considered in all basal pneumonic processes of undetermined etiology. Chief differentiation is when the lesion (paraffinoma) resembles neoplasm. Clinical, radiologic, and cytologic approach is necessary for establishing a diagnosis.

6. Aspiration of gastric contents produces pulmonary edema early and

may cause acute bronchial obstruction. Later foreign body reaction occurs and leads to chronic bronchial obstruction and pulmonary fibrosis.

7. Pathologic changes in the lung after aspiration of a foreign body depend on site of lodgements, chemical and physical characteristics of the substance, and length of time that elapses before its removal.

8. There is a whole range of aspiration pneumonias due to inhalation of infected material from the mouth and throat varying in severity from solitary lung abscess and a predominant pneumonic process, to less severe forms of respiratory tract infections.

9. Aspiration of infected material from the mouth and throat occurs in patients with poor dental hygiene, with poor or absent gag reflexes due to states of unconsciousness, most commonly seen in alcoholics.

RESUMEN

1. La neumonía por aspiración es un problema diagnóstico que puede ser difícil y que frecuentemente es pasado por alto en el diagnóstico diferencial.

2. Las neumonías por aspiración pueden resultar por: (a) aspiración de aceites o lipoides, (b) alimentos o secreciones gástricas, (c) cuerpos extraños, y (d) materiales infectados.

3. Se presentan casos ejemplares de cada forma.

4. Las neumonías por aspiración tienen tendencia a localizarse en las partes más declives en el momento de la aspiración.

5. La neumonía lipoidica debe considerarse en los procesos neumónicos basales de etiología indeterminada. La diferenciación principal es la lesión que se parece a una neoplasia (parafinoma).

Los estudios clínico, radiológico, y citológico se requieren para establecer el diagnóstico.

6. La aspiración del contenido gástrico produce al principio edema y puede producir obstrucción bronquial aguda. Después hay reacción de cuerpo extraño que conduce a obstrucción bronquial crónico y fibrosis pulmonar.

7. Los cambios patológicos después de la aspiración de cuerpos extraños dependen del lugar en que se alojan de las características químicas y físicas de las substancias y del tiempo transcurrido antes de su extracción.

8. Hay toda una variedad de neumonías de aspiración debidas a la inhalación de material infectado de la boca y de la garganta variando en gravedad desde el abscesso solitario y los procesos de predominio neumónico hasta las menos severas formas después de la infección de las vías respiratorias.

9. La aspiración de material infectado a partir de la boca y de la garganta ocurren en enfermos con mala higiene bucal, con malos reflejos nauseosos y ausencia de ellos, debido a estados de inconsciencia, comunmente más vistos en los alcohólicos.

RESUME

1. La pneumonie par aspiration est un problème de pneumologie qui peut présenter beaucoup de difficultés diagnostiques et est fréquemment négligé dans le diagnostic différentiel des lésions pulmonaires.

2. Les pneumonies par aspiration peuvent résulter de: a) aspiration de matières huileuses, b) nourriture et sécrétions gastriques, c) corps étrangers, d) matériaux infectés.

3. Les auteurs présentent des cas illustrés de chacun de ces types.

4. Les pneumonies par aspiration ont une tendance à la localisation dans les zones les plus facilement accessibles des poumons au moment de l'aspiration.

5. Les pneumonies huileuses devraient être considérées comme des processus pneumoniques liés à la présence d'huile d'étiologie indéterminée. La difficulté principale du diagnostic se présente lorsque la lésion (paraffinome) ressemble à une néoplasie. Des examens cliniques, radiologiques et cytologiques sont nécessaires pour établir le diagnostic.

6. L'aspiration des produits gastriques produit un oedème pulmonaire précoce et peut provoquer une obstruction bronchique aiguë. Plus tard se constitue un syndrome de corps étranger intrabronchique qui entraine une obstruction bronchique chronique avec fibrose pulmonaire.

7. Les altérations anatomo-pathologiques du poumon après aspiration d'un corps étranger dépendent du siège des localisations, des caractéristiques chimiques et physiques de la substance inhalée, et du temps qui s'est écoulé entre le début de l'aspiration et sa cessation.

8. Il y a une série complète de pneumonies par aspiration dues à l'inhalation de matériaux infectés, par la bouche et la gorge, qui varient en gravité depuis l'abcès pulmonaire isolé ou le processus pneumonique prédominant jusqu'à des manifestations moins graves consécutives à l'infection de l'appareil respiratoire.

9. L'aspiration de matériel infecté par la bouche et la gorge survient chez les malades qui ont une mauvaise hygiène dentaire et dont les réflexes sont atténués ou absents. Ceci est secondaire à un état d'inconscience, que l'on constate le plus souvent chez les alcooliques.

ZUSAMMENFASSUNG

1. Die Aspirations-Pneumonie ist ein die Lunge betreffendes Problem, das ein erhebliches Mass an diagnostischen Schwierigkeiten bieten kann und häufig vernachlässigt wird bei der Differential-Diagnose pulmonaler Veränderungen.

2. Aspirations-Pneumonien können entstehen durch (a) Aspiration öliger Fette, (b) Sekret von Nahrung und aus dem Magen, (c) Fremdkörper und (d) Infiziertes Material.

3. Zur Veranschaulichung werden Fälle eines jeden Typs vorgewiesen.

4. Aspirations-Pneumonien haben eine Tendenz zur Lokalisation indem zum Zeitpunkt der Aspiration am meisten abhängigen Lungenabschnitt.

5. Lipoid-Pneumonien muss man zunächst als pneumonische Prozesse unbestimmter Herkunft ansehen. Die Differenzierung ist besonders notwendig, wenn der Befund (Paraffinom) einer Neubildung ähnlich sieht. Ein Vorstoss in klinischer, röntgenologischer und cytologischer Hinsicht ist erforderlich, um zu einer Diagnose zu kommen.

6. Aspiration von Mageninhalt macht frühzeitig ein Lungenödem und kann zu akutem Bronchialverschluss führen. Später tritt dann eine Fremdkörperreaktion ein und führt zu chronischem Bronchialverschluss und einer Lungenfibrose.

7. Die pathologischen Veränderungen in der Lunge nach Fremdkörperaspiration hängen von der Region der Ausbreitung ab, ferner von den chemischen und physikalischen Eigenheiten der Substanz, sowie der Zeitspanne, die bis zu ihrer Entfernung verstreicht.

8. Es besteht ein weiter Spielraum bei den Aspirations-Pneumonien infolge Inhalation infizierten Materials aus Mund und Rachen, die in ihrer Schwere variieren vom einfachen Lungenabszess und überwiegend pneumonischen Prozess bis zu weniger schweren Formen im Anschluss an Infektionen des Respirationstraktes.

9. Aspiration von infiziertem Material aus Mund und Rachen tritt auf bei Patienten mit schlechten Zahnverhältnissen, mit mangelhaftem oder fehlenden Würgreflex im Zusammenhang mit Zuständen von Bewusstlosigkeit, wie man sie häufig bei Alkoholikern sieht.

Complete reference list will appear in reprints

Role of Photofluorography in Navy Tuberculosis Control*

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The primary goal of the Tuberculosis Control Program in the Navy is to prevent dissemination of this communicable disease through detection of personnel suffering from tuberculosis in its earliest stages. Thus, the exposure of healthy associates to the disease is minimized and treatment of these individuals can be accomplished at a time when the best results can be obtained. Periodic evaluation of the effectiveness of the program, which has been described elsewhere,[1] is necessary to ensure that this objective is being obtained to a degree that makes continuation of the program economically and medically worthwhile. The following study was made to find out how effective a contribution routine photofluorographic screening makes to the program. Primary interest lies in the periodic or annual photofluorogram.

The pilot study reported herein, which utilized a sampling technique, was considered to be one method of evaluating this program. The particular objective was to determine the percentage of cases of established tuberculosis brought under treatment as a result of original detection by routine photofluorographic procedures, this being the principal case finding method applicable to the Navy and Marine Corps as a whole. The assumption is made that detection by photofluorographic screening resulted in early isolation and therapy of cases which might have progressed for some time before symptoms or some other illness would have resulted in discovery of their disease. Thus a more prolonged exposure of infected personnel to their associates was prevented.

The study had the following specific objectives:

1. To determine the proportion of total cases of tuberculosis occupying beds at the two special treatment hospitals during the year 1955 which had originally been detected by photofluorographic screening.

2. To determine what proportion of the cases discovered by photofluorography were detected by routine *periodic* 70 mm. photofluorography.

3. To determine the actual time spread between periodic examinations in the cases reviewed.

4. To determine the proportion of cases of tuberculosis revealed only in a second reading of the photofluorograms.

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Although the mechanism of operation of the tuberculosis control program in the Navy has been described in an earlier paper,[1] it seems appropriate to review some of the pertinent details in relation to the present study. Routine chest roentgenographic examinations are made of both military and civilian personnel upon their entering service, upon their separation from service, and at annual intervals when practicable. Survey x-ray inspections are taken in almost all instances by 70-mm. photofluorographic technique. An immediate interpretation of the film is made at the various stations, and 14 by 17 inch films are ordered for those individuals whose 70-mm. films reveal suspicious findings. If the suspicious area is verified by the 14 by 17 inch film, the service man is sent to a naval hospital for consultation or admission as appropriate. Each month all 70-mm. films that have been taken are mailed to the Tuberculosis Control Section of the Navy Bureau of Medicine and Surgery in Washington, D. C., where they are again reviewed. The films are accompanied by a log that gives adequate identification of each film by the interpreting physician. If a 70-mm. film that was interpreted as negative in the field is found to be suspicious in the course of re-reading, a request for follow-up is sent to the activity where the individual is stationed. Personnel with chest disease suggested of being tuberculous are isolated, studied and treated in United States naval hospitals with further transfer of such patients to the two established pulmonary diagnostic and treatment centers at St. Albans, Long-Island, New York, and San Diego, California.

Methodology

The two special treatment hospitals for tuberculosis mentioned above were requested to provide a special quarterly report on all patients being studied on the wards of the Chest Services on May 31, 1955; August 31, 1955; November 30, 1955; and February 29, 1956. The reports consisted of the following information on each patient: (1) name; (2) rank or rate; (3) serial number; (4) tentative diagnosis; and (5) reason for admission.

The reason for admission was divided into three main categories: (1) admitted because of symptoms referable to possible pulmonary disease; (2) because of suspicious routine 70-mm. photofluorogram; and (3) bebecause of unrelated medical reason with the pathological pulmonary condition subsequently discovered.

Admission resulting from routine 70-mm. chest photofluorographic surveys were further subdivided into the following categories: (a) routine enlistment film taken on induction, enlistment, or recall to active duty; (b) re-enlistment for extension of active duty; (c) officer annual physical examinations required each year, of which the chest film is an integral part; (d) separation films required immediately before release from active duty or retirement; and (e) periodic films taken annually, when possible, on all active duty personnel.

With the information provided by the quarterly reports from the designated U. S. naval hospitals, searches were made in the Bureau of Medicine and Surgery for the final diagnoses on these selected cases on Individual Statistical Report of patient (NAVMED-F) cards, which are made out on discharge of all patients from Naval Hospitals. All cases in which the final diagnosis was inactive tuberculosis or of non-tuberculous or unknown

TABLE I
NUMBER OF NEW CASES OF ACTIVE PULMONARY TUBERCULOSIS REVIEWED WITH REASONS FOR ADMISSION TO NAVAL HOSPITALS

Reason for Admission	Number	Per Cent
Symptoms	111	37
Routine 70-mm. photofluorograph	168	56
Other	22	7
TOTAL	301	100

etiology were eliminated from the study. This left only established cases of active pulmonary and pleural tuberculosis (according to National Tuberculosis Association Standards, 1955), in the study.

The medical record jackets corresponding to the individual names and service numbers of these patients were procured and carefully reviewed to obtain information regarding the interval in months between the last routine x-ray film and the one which demonstrated pathology; and also to determine whether or not each individual had received an x-ray film when he came on active duty.

Finally, the completed list was checked against the Tuberculosis Control files to determine if the patient had been discovered by review of his film (the initial reading in the field having been interpreted as negative or within normal limits). Twenty-one cases in which medical records were either not available or incomplete were discarded from the study, leaving the final total of cases at 301, or approximately a 90 per cent sample of all new cases (329) of active pulmonary tuberculosis discovered in the Navy during this period.[2] The omission of these 21 cases with incomplete information did not materially effect the result since tabulations made including these cases were not significantly different from the data presented herein.

Results

Table I shows the total number of cases reviewed and the reasons for their admission to hospitals. They are classified as to whether the patients were admitted because of symptoms referable to chest conditions; or were discovered by use of 70-mm. photofluorograms; or because of unrelated conditions, and later found to have tuberculosis.

Table II classifies the patients who were admitted because of routine 70-mm. photofluorographs into separate categories according to reasons

TABLE II
CASES OF ACTIVE PULMONARY TUBERCULOSIS DISCOVERED BY 70-MM. PHOTOFLUOROGRAMS

Reason for Photofluorogram	Number	Per Cent
Enlistment	24	14
Separation	31	18
Re-enlistment	11	7
Periodic	102	61
TOTAL	168	100

TABLE III
TIME INTERVAL, IN MONTHS, SINCE LAST CHEST FILM WAS CONDUCTED ACCORDING TO REASONS FOR ADMISSION TO NAVAL HOSPITALS

Reason for Admission		Total Cases	Time in Months Since Last X-Ray				
			0	1-11	12-17	18-23	over 24
Symptoms		111	0	60	22	12	17
Others		22	0	12	5	3	2
70-mm. photofluorograph	All photofluorographs	168	23	70	33	20	22
	Periodic	102	0	47	24	15	16
	Enlistment	24	23	1*	0	0	0
	Re-enlistment	11	0	7	2	0	2
	Separation	31	0	15	7	5	4
TOTAL		301	23	142	60	35	41

*An enlistment film that was initially positive, but was not discovered until a review of the film sometime later in the Bureau.

for the photofluorograph. These are subdivided into entrance examinations, separation examinations, re-enlistment examinations, and routine periodic examinations.

The routine annual periodic examination of all personnel on active duty was responsible for 61 per cent of all cases discovered by 70-mm. photofluorography. Also, of the 301 cases reviewed, it was responsible for 34 per cent of the total. It is of interest to note that 24 cases of active pulmonary tuberculosis were discovered in enlistment films, which is 8 per cent of the total cases reviewed. Some of these individuals had previously been examined by a chest x-ray film before coming on active duty, and their films interpreted as negative. Others failed to have a preinduction film taken due to lack of facilities or for other reasons.

Table III reveals the time interval in each of the three main groups since the last film was taken, with a further breakdown of subdivisions under 70-mm. film.

Of the total of 301 cases reviewed, only 41 (14 per cent) had not been x-rayed within a 24 month period. In 285 (75 per cent) of these cases a chest x-ray film had been taken within the preceding 18 months which was interpreted as "negative" or the findings were not considered to be of clinical significance and compatible with return to duty status.

Table IV is a compilation of the results of second reading of films at the Bureau of Medicine and Surgery. The value of such a second reading has

TABLE IV
TOTAL NEW CASES OF PULMONARY TUBERCULOSIS DISCOVERED BY 70-MM. PHOTOFLUOROGRAPHY SHOWING CASES DISCOVERED IN THE FIELD AND AT THE BUREAU OF MEDICINE AND SURGERY

	Number	Per Cent
Total cases discovered by 70-mm.	168	100
Discovered initially in the field	150	89
Discovered initially in the Bureau	18	11

been proved many times,[3,4] but has always been of interest in a strict analysis of how many actual cases of active pulmonary tuberculosis were being discovered by this review.

Of the 168 cases discovered by the 70-mm. photofluorography, 18 (11 per cent) initially interpreted as negative or within normal limits were later proved to show active tuberculosis as a direct result of film review.

Discussion

The method described in this study was selected for several reasons. In the first place, the data desired for analysis is not always incorporated in medical records, but could be obtained in all cases in this study by direct interrogation of the hospitalized patient. Secondly, because less than 1 per cent of individuals reported to have abnormal chest x-ray films are finally shown to have active pulmonary tuberculosis, such a sample analysis obviated the unfruitful search of many records later found to be unsuitable for analysis. Thirdly, since patients with chronic pulmonary disease are generally under observation and treatment at Navy Tuberculosis Centers for an average of three or four months, the majority of pulmonary tuberculosis cases discovered in the Navy and Marine Corps through the year could thus be personally interviewed when quarterly roster reports were made out. The final tabulations showed this to be true as the cases reported represented approximately 90 per cent of all new cases of active pulmonary tuberculosis discovered in the Navy during this period.[10]

Anderson[5] has said that periodic x-ray film examinations of healthy people is necessary to keep the prevalence of tuberculosis at a low level until better methods are found. Britten and Charter,[6] in a previous study of Naval and Marine Corps personnel, pointed out that a large number of cases of active tuberculosis were discovered in personnel after years of active service, attesting to the need for periodic chest x-ray film examinations of all personnel over a long period. Other reports of Navy surveys[7-10] have also indicated the value of photofluoroscopic chest surveys in the U. S. Navy but none previously have demonstrated how many established cases of tuberculosis are actually discovered initially by the routine periodic x-ray film.

No significant downward trend in the annual number of cases of suspected tuberculosis has been noted in this program. Tuberculosis, in all forms, was the fifth leading cause of invaliding from the U. S. Naval Service during 1955.[2] It is well known that the mortality rate from tuberculosis has declined steadily since the first of the century. Although this drop has been almost precipitous since the advent of chemotherapy and resectional surgery during the past 10 years, Waring and others have pointed out that unfortunately the rate of new cases reported has not shown as marked a fall.[11,12]

The fact that 18 (11 per cent) of the 168 cases discovered for all reasons by 70-mm. photofluorograms, were revealed by a second reading does not in any sense of the word cast unfavorable reflection on the ability of radiologists or others to detect tuberculosis, but simply notes a percentage of human error which is consistent with similar results reported in the studies of Groth-Petersen, Lovgreen, and Thillemann,[3] as well as those of Long, Stein, and Henderson.[4]

The discovery of 24 cases of active tuberculosis among recruits at induction appears to reemphasize the need for preinduction films and the necessity for careful interpretation of them. The importance of this in enabling the Navy and Marine Corps to exclude infected personnel from possible contact with healthy individuals at training centers, and to minimize the number of disability pensions is recognized. It would seem that this is an area of control as yet incompletely solved.

A later paper will break down the extent of disease in each of these cases in order to discover if there was any correlation between the amount of disease present and the length of time since the last chest film, such correlation having been noted by Anderson et al., in their study of x-ray surveys.[13]

SUMMARY

A review of 301 newly discovered cases of active pulmonary tuberculosis among Navy and Marine Corps Personnel during 1955 was made. This represented approximately a 90 per cent sample of all such cases discovered during this period. The reasons for hospitalization of these patients included 111 cases (37 per cent) admitted because of symptoms; and 22 cases (7 per cent) discovered to have pulmonary disease after admission to the hospital for unrelated conditions. Of the 168 cases admitted because of abnormal 70-mm. photofluorograms, 102 (61 per cent) were discovered by routine periodic annual examinations as contrasted to 66 (39 per cent) detected at the time of enlistment, re-enlistment into, or separation from the service. A review, or second reading, of all films, which is accomplished routinely, accounted for 18 of these 168 cases.

It has thus been shown in the present study that the majority of new cases of tuberculosis initially diagnosed in the U. S. Navy are discovered by means of photofluorography, and the majority of such cases are detected by the periodic (annual) chest x-ray film.

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RESUMEN

Se hizo un estudio de 301 casos nuevos de tuberculosis descubiertos durante 1955 en el personal del Cuerpo de Marina de los Estados Unidos. Esto representa una muestra equivalente al 90 por ciento de tales casos descubiertos en ese período.

Las razones para la hospitalización de estos enfermos fueron: 111 (37 por ciento) admitidos por tener síntomas; y 22 casos (7 por ciento) se descubrió que tenían tuberculosis después de ingresar al hospital por causas sin relación con la enfermedad descubierta incidentalmente. De los 168 admitidos por tener una roentgenfotografía anormal (70 mm.), 102 (61 por ciento) se descubrieron por los exámenes anuales de rutina en contraste con 66 (39 por ciento) descubiertos al alistarse o al re-alistarse o bien al separarse del servicio. Una revisión de segunda lectura de todas las películas, lo que se hace habitualmente, permitió encontrar 18 de estos 168 casos.

Así se ha demostrado por el estudio actual que la mayoría de los casos nuevos de tuberculosis diagnosticados en la Marina de los Estados Unidos se descubren por la roentgenfotografía y que la mayoría de tales casos son evidenciados por el examen periódico anual por la película radiológica.

RESUME

L'auteur a fait l'étude de 301 cas de tuberculose pulmonaire évolutive récemment découverts chez le personnel de la "Navy and Marine Corps" pendant l'année 1955. Ceci représente environ 90% de tous les cas ainsi découverts pendant cette période. On se basa pour décider l'hospitalisation sur les symptômes. Ainsi furent hospitalisés III cas (37%). 22 cas furent découverts porteurs d'affection pulmonaire après admission à l'Hôpital pour une raison non précisée. Sur les 168 cas admis à cause d'une radiophotographie anormale, 102 (61%) furent découverts par les examens systématiques annuels, alors que 66 (39%) furent dépistés au moment de l'embauche, de la ré-embauche ou du renvoi du service. Une revue, ou seconde lecture, de tous les films, qui fut pratiquée d'une façon systématique, fit connaitre 18 des 168 cas précédents.

La présente étude montre que la majorité des nouveaux cas de tuberculose initialement diagnostiquée dans la Marine sont découverts au moyen de la radiophotographie, et que la majorité de ces cas sont dépistés par les examens radiologiques annuels systématiques.

ZUSAMMENFASSUNG

Es wurden 3001 frisch entdeckte Fälle von aktiver Lungentuberkulose unter dem Personal des Navy- und Marine-Corps während des Jahres 1955 einer Durchsicht unterzogen. Diese stellten ein Muster von ungefähr 90% von allen während dieser Zeit entdeckten Fälle dar. Die Gründe für die Krankenhauseinweisung dieser Patienten umfasste 111 Fälle = 37%, die Wegen Symptomen zugewiesen wurden, sowie 22 Fälle = 7%, bei denen man nach Einweisung in Krankenhaus feststellte, dass sie eine Lungenerkrankung hatten; sie wurden aus nicht in Beziehung stehenden Gründen überwiesen. Von den 168 wegen krankhafter 70mm-Schirmbilder überwiesenen Fälle warem 102 (61%) entdekt worden durch routinemässige periodische jährliche Untersuchungen im Gegensatz zu 66 Fällen (39%), deren Befunde bemerkt wurden z. Z. der Musterung, erneuten Musterung zum oder Entlassung aus dem aktiven Dienst. Eine Durchsicht oder eine 2. Befunderhebung in allen Fällen, wie sie routinemässig durchgeführt wird, war für 18 dieser 168 Fälle die Ursache.

Es wurde auf diese Weise durch die vorliegende Untersuchung gezeigt, dass die Mehrzahl der neuen Tuberkulosefälle, die zuerst in der Marine der USA erkannt werden, mit Hilfe der Schirmbildfotografie festgestellt werden; und die Mehrzahl solcher Fälle werden erkannt durch periodische (jährliche) Thorax-Röntgenaufnahmen.

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The Decreasing Hazard of Surgical Procedures on Patients with Asthma*

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Asthma is a common and serious disease. As it is often necessary to subject patients with asthma to major or minor surgical procedures, it is important to know how safely such measures can be carried out.

The major purpose of this paper is to contrast the incidence of pulmonary complications observed in two earlier series with the incidence observed in a series of 153 patients with asthma who underwent major surgical procedures at the Mayo Clinic in the period January, 1950, through December, 1955, during an era when effective antibiotics were available. Pulmonary complications encountered in all three groups consisted principally of severe asthma, bronchopneumonia and obstructive pneumonitis. A number of patients in all three groups experienced very mild asthma postoperatively, but these patients were not classified as having pulmonary complications. Only severe status asthmaticus or prolonged asthmatic attacks were included as pulmonary complications.

A secondary purpose of the paper is to report on the incidence of pulmonary complications in an additional 178 patients who underwent minor operations.

Previous Studies

Two studies similar to the present one have been made previously at the clinic. The first,[1] reported in 1942, included 189 consecutive patients with asthma who underwent major surgical procedures in the period July, 1934, through September, 1940. The second study,[2] reported in 1944, included 142 consecutive patients with asthma who underwent major surgical operations in the period October, 1940, through July, 1943. Both previous papers were published before the widespread use of antibiotics.

In the first series, 25 of the 189 patients (13.2 per cent) had some type of pulmonary complication. Pneumonia developed in 10 patients, severe asthma in nine, bronchitis in three, obstructive pneumonitis in two and pulmonary infarction in one. Four postoperative deaths occurred, giving a mortality rate of 2.1 per cent; two of the deaths were related to pulmonary complications (severe asthma and bronchopneumonia) and were presumably responsible, to a great extent, for the subsequent deaths. Approximately 87 per cent of the patients, therefore, underwent operation without pulmonary complications.

In the second series, the study of which was completed during the early years of sulfonamide therapy, 22 of 142 patients (15 per cent) experienced some pulmonary complication. Bronchopneumonia developed in six, obstructive pneumonitis in six, bronchitis in six and severe asthma in four.

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Two postoperative deaths occurred, giving a mortality rate of 1.4 per cent; both deaths followed pneumonia. Thus, 85 per cent of this group of patients with asthma underwent operation without pulmonary complications.

Present Study

Patients Who Underwent Major Operations.—Some type of pulmonary complication developed postoperatively in only six (3.9 per cent) of the current series of 153 asthmatic patients who underwent major operations. Two of the six patients had bronchopneumonia, two had obstructive pneumonitis, and two had severe asthma (table). This incidence of 3.9 per cent is significantly less than the incidence of 13.2 per cent and 15 per cent respectively for the previous series.

There were two deaths, giving a mortality rate of 1.3 per cent. Bron-

PULMONARY COMPLICATIONS ASSOCIATED WITH OPERATIONS ON ASTHMATIC PATIENTS

Site or Operation	Total Patients	Patients with Complications				
		Obstructive Pneumonitis	Broncho-pneumonia	Asthma	Total	Deaths
Major Procedures						
Gallbladder	33	1		1	2	
Stomach and duodenum	15		1		1	1
Colon	11		1	1	2	1
Hernia	16					
Pelvis	16	1			1	
Prostate	13					
Thyroid	22					
Breast	6					
Thorax	3					
Miscellaneous	18					
TOTAL	153	2 (1.3%)	2 (1.3%)	2 (1.3%)	6 (3.9%)	2 (1.3%)
Minor Procedures						
Bronchoscopy	81			4	4	1
Intranasal	50					
Esophagoscopy	4					
Tonsillectomy and adenoidectomy	4					
Extraction of teeth	4					
Dilatation and curettage	11					
Miscellaneous	24					
TOTAL	178			4 (2.2%)	4 (2.2%)	1 (0.6%)

chopneumonia appeared to be the cause of death in a patient 77 years of age who had long-standing chronic asthmatic bronchitis and an emphysematous barrel chest with secondary polycythemia. The patient underwent an exploratory operation because of an obstructing prepyloric lesion (visualized roentgenographically) and a duodenal ulcer that penetrated the pancreas. Posterior gastroenterostomy and pyloroplasty for pyloric hypertrophy were performed.

The other death was attributed to severe asthma which came on postoperatively 4 days before death. This patient, a man 64 years old, had been a heavy smoker for 40 years and had chronic bronchitis and dyspnea on exertion, but gave no history of having had paroxysmal nocturnal asthma preoperatively. His operation consisted of palliative segmental resection for a carcinoma of the sigmoid that had spread to the liver.

Patients Who Underwent Minor Operations.—The records of 178 asthmatic patients who underwent minor operations during the period mentioned were also reviewed. Four of 81 patients had severe asthma following bronchoscopy, which was the only minor surgical procedure that was followed by a pulmonary complication; thus, 2.2 per cent of the 178 patients had such a complication. Cardiac arrest following bronchoscopy resulted in one death. The arrest was thought to be secondary to severe bronchospasm. Fifty intranasal operations of various types were accomplished without a single pulmonary complication. The remaining 47 miscellaneous minor surgical procedures were performed without incident.

Combined Groups.—In the combined groups, which totalled 331 patients, antibiotics were administered pre-operatively to 69 patients and postoperatively to 112. There were altogether 10 patients who had pulmonary complications, of whom seven patients were classed as having the intrinsic type of asthma and three as a combination of intrinsic and extrinsic asthma. In no instance was asthma due wholly to extrinsic factors. Four patients who had complications received antibiotics preoperatively and six received them postoperatively. Steroids were administered postoperatively in three cases. Seven of those who had complications were women and three were men.

The average age of the 331 patients was 54 years, the oldest being 77 years and the youngest 32. The average duration of asthmatic symptoms was 10 years, the longest being 25 years and the shortest two years.

General anesthetic agents consisted mainly of ether or intravenously administered thiopental (pentothal) sodium. Topical anesthesia was used for all intranasal operations and bronchscopic procedures. No untoward reactions were attributed to the anesthetic agents.

In the two previous papers the incidence of postoperative pulmonary complications was reported to be significantly increased after operations in the upper part of the abdomen, presumably owing to splinting of the diaphragm and the lower thoracic muscles. This observation also appears to be true of the present series, although the data are meager.

It is not possible to compare mortality rates in the three series, because of the variety of underlying diseases present other than bronchial asthma. It is of interest, however, to note that the mortality rate thought to be attributable to pulmonary complications is approximately the same in the three groups (2.1, 1.4 and 1.3 per cent respectively).

Means of Reducing Surgical Hazards

It would be an obvious error to attribute this favorable drop in the frequency of postoperative pulmonary complications to the use of antibiotics alone. Other factors that have contributed to lowering of such complications include careful selection and preoperative preparation of patients, scheduling of the operation at a time most favorable for anesthesia and surgical treatment, postoperative measures to assure an unobstructed airway, and the use of steroid therapy if indicated.

Thus, patients with acute or subsiding respiratory infections and asthma have been denied operation until the infection has entirely subsided. Asthmatic patients who have required general anesthesia have been hospitalized for at least two nights of observation to determine their ability to lie flat in bed (one pillow) all night without having cough or asthma. Appropriate therapy has been administered as long as necessary in the hospital to attain this important objective before submitting any patient to anesthesia and operation. Preoperative rest, humidification of the room, adequate oral and intravenous hydration, nebulization of antibiotics, alevaire and sympathomimetic medications, and croup tents, face tents and oxygen are among the agents or measures that have been used. Epinephrine or norepinephrine, theophylline preparations intravenously and rectally administered, and antihistamines for sedation have been used commonly as occasion has demanded.

Also much has been accomplished in the instruction of resident and nursing personnel in the prevention of pulmonary complications after operation. The necessity of moving the patient frequently in the immediate postoperative period has been emphasized to all concerned, as well as the importance of limiting sedatives and narcotics to a bare minimum. The necessity for the use of suction or bronchoscopy for the removal of tracheobronchial secretions has been materially lessened by these precautions. The intermittent inhalation of carbon dioxide to promote better inflation of the lungs has been used when indicated. When asthma is complicated by emphysema, serious mechanical problems exist that require particular caution, even to denying any but lifesaving operations.

There can be no question that improved technics in the management of asthma together with a better understanding of the underlying disease have contributed to the prevention of postoperative complications.

SUMMARY

In a series of 153 patients with asthma who underwent major surgical procedures at the Mayo Clinic from January, 1950, through December, 1955, there were six (3.9 per cent) who had pulmonary complications after operation. This percentage is significantly less than the 13.2 per cent and 15 per cent respectively observed in two previously reported series from the clinic prior to the widespread use of antibiotics. The decrease is attributed mainly to the optimal control of asthma before and after operation, to the use of antibiotics when indicated, and to the newer steroid therapy. Thus, 96.1 per cent of these patients underwent major surgical procedures without the development of significant postoperative pulmonary complications. It is concluded that the properly prepared asthmatic patient is a good surgical risk.

Pulmonary complications occurred in four of 178 asthmatic patients (2.2 per cent) who underwent minor operations. All the complications consisted of severe asthma and all were associated with bronchoscopy. There was one death in this category.

The mortality rates for the two previously reported series and for the present series of 153 patients who underwent major operations were approximately the same (2.1, 1.4 and 1.3 per cent respectively). It is impossible, however, to compare these rates statistically because of the variety of diseases present other than bronchial asthma.

The majority of complications occurred in those patients with intrinsic asthma of long duration.

No untoward effects were attributed to the anesthetic agents used.

RESUMEN

En una serie de 153 enfermos de asma que se sujetaron a cirugía mayor en la Clínica Mayo de Enero de 1950 hasta Dic. de 1955, hubo 6 (3.9 por ciento) que tuvieron complicaciones pulmonares después de la operación. Este porcentaje es significantemente menor que el 13.2 por ciento y el 15 por ciento respectivamente, referidos en dos series antes observadas en la Clínica antes del advenimiento de los antibióticos. El decrecimiento se atribuye principalmente al óptimo control del asma de la operación, al uso de los antibióticos cuando están indicados y a la nueva terapéutica por los esteroides. Así, 96.1 por ciento de estos enfermos pasaron por operaciones mayores sin que se desarrollaran complicaciones pulmonares de importancia. De ahí se concluye que el asmático debidamente preparado es un buen riesgo operatorio.

Ocurrieron complicaciones pulmonares en cuatro de 178 asmáticos (2.2 por ciento) en operaciones menores. Todas las complicaciones consistieron en asma severa y todas estaban asociadas con la broncoscopía. En este grupo hubo una muerte.

La mortalidad para las dos series antes referidas y para las actuales, de 153 enfermos que sufrieron cirugía mayor, fué aproximadamente la misma (2.1, 1.4 y 1.3 respectivamente). Sin embargo, es imposible comparar estas cifras estadísticamente a causa de la variedad de enfermedades, aparte del asma, que presentaban.

La mayoría de las complicaciones ocurrieron en enfermos con asma intrincada de larga duración.

No se atribuyeron efectos nocivos a los anestésicos usados.

RESUME

Dans un groupe de 153 malades, atteints d'asthme, qui subirent des interventions chirurgicales importantes à la Clinique Mayo, de Janvier 1950 à Décembre 1955, six (3,9%) ont présenté des complications pulmonaires post-opératoires. Ce pourcentage est infiniment moindre que celui de 13,2% et de 15% respectivement observés dans deux groupes rapportés antérieurement, alors que l'emploi des antibiotiques n'était pas encore étendu. La décroissance est attribuée principalement au fait que l'on peut mieux lutter contre l'asthme avant et après l'opération, à l'emploi des antibiotiques lorsqu'ils sont indiqués, et à la très récente corticothérapie. Ce sont les raisons qui ont permis à 96,1% de ces malades de subir des inter-

ventions chirurgicales importantes sans complications pulmonaires post-opératoires importantes. L'auteur en conclut que l'asthmatique convenablement préparé ne court pas de risques chirurgicaux particuliers.

Des complications pulmonaires survinrent chez quatre asthmatiques sur 178 (2,2%) qui subirent des interventions chirurgicales mineures. Toutes les complications consistèrent en asthme sévère et toutes firent suite à une bronchoscopie. Il y eut une mort dans cette catégorie.

Les taux de mortalité pour les deux groupes antérieurement rapportés et pour le présent groupe de 153 malades qui subirent de grandes interventions, furent approximativement les mêmes (2,1; 1,4 et 1,3 respectivement). Il est impossible cependant, de comparer statistiquement ces taux à cause de la variété des affections traitées chez les asthmatiques.

La majorité des complications survint chez les malades atteints d'asthme durant depuis longtemps.

On ne peut attribuer un rôle dans les complications aux agents anesthétiques qui furent utilisés.

ZUSAMMENFASSUNG

In einer Reihe von 153 Patienten mit Asthma, die grösseren chirurgischen Eingriffen unterzogen wurden an der Mayo-Klinik von Januar 1950 bis Ende Dezember 1955, kamen 6 Fälle vor (3,9%), bei denen pulomonale Komplikationen auftraten nach der Operation. Dieser %-Satz ist entscheidend geringer als die 13,2% bzw. 15%, die in 2 zuvor veröffentlichten Serien von der Klinik mitgeteilt worden waren vor der verbreiteten Anwendung von Antibioticis. Die Verringerung ist in erster Linie der optimalen Kontrolle des Asthmas vor und nach der Operation zuzuschreiben, ferner dem Einsatz von Antibioticis, wenn diese indiziert waren, so wie der neueren Steroid-Therapie. So erfolgten bei 96,1% dieser Kranken grössere chirurgische Eingriffe ohne Entwicklung beträchtlicher Post-operativer Lungenkomplikationen. Es wird daraus der Schluss gezogen, dass bei dementsprechend vorbereiteten Asthmatiker eine Operation kein besonderes Risiko bedeutet.

Pulmonale Komplikationen traten bei 4 von 178 asthmatischen Kranken auf (2,2%), bei denen kleinere Operationen vorgenommen wurden. Diese Komplikationen bestanden sämtlich in schwerem Asthma und alle kamen vor bei Bronchoskopien. In dieser Kategorie trat auch 1 Todesfall ein.

Die Mortalitätsziffern der beiden zuvor mitgeteilten Serien und für die gegenwärtige Serie von 153 Kranken, bei denen grössere Operationen vorgenommen wurden, waren ungefähr die gleichen (2,1%; 1,4% bzw. 1,3%). Es ist jedoch unmöglich, diese Werte statistisch zu vergleichen im Hinblick auf die Vielzahl von Erkrankungen, die abgesehen vom Bronchialasthma noch vorlagen.

Die Mehrzahl der Komplikationen trat bei Kranken mit echtem Asthma von langer Dauer ein.

Den angewandten Ansthaeticis zuzuschreibende ungünstige Nebenwirkungen wurden nicht beobachtet.

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Tuberculin in Diagnosis Past, Present and Future*

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It has been refreshing and encouraging to observe you, a group of 200 physicians in general practice, enter the tuberculosis field in 1952 and continue without prejudice, preconceived ideas, etc., etc., and strike mighty blows at the very heart of tuberculosis. You have not wasted valuable time and effort on speculations, theories or anachronisms. Instead you waived all such items aside in the beginning and went forth with the most powerful weapons yet devised for the eradication of tuberculosis.

This disease had plagued the world since prehistoric times, and no hope of controlling and eventually eradicating it was seen until Koch isolated the tubercle bacillus in 1882 and produced tuberculin in 1890. Soon thereafter, veterinarians in Germany, Russia, Denmark, the United States, and other nations conducted large studies to determine its specificity and accuracy in the diagnosis of tuberculosis in animals.

In 1891 Gutmann, Dorpat, Russia, tested a large number of cattle and at autopsy found that practically all reactors possessed lesions, but nonreactors were nearly always free from them. Bang, State Veterinarian in Denmark, initiated studies about the same time. Concerning this work, he later said the degree of reaction in cattle had no constant relation to the extent of disease: "Farmers often believe the cow reacting violently to be extensively tuberculous and one reacting feebly to be comparatively sound. The case is rather the reverse, but this is not constant either." After an extensive testing and post mortem experience, he said he had known only three or four cases in which he could find no tuberculous deposit in reactor animals: "In these cases the fault may be mine and not that of tuberculin as it is impossible to examine every part of the body of a slaughtered animal. The tubercles may have been hidden in an unusual place or they may have been so small or so fresh that they could not be discovered." About this he said, "Moreover, it must be considered that it would not be a disaster even if a sound animal should for once be considered tuberculous." He observed in some nonreactors "very small," "very old," or "greatly calcified" lesions indicating that the process had become stationary. Thus, he may have described for the first time marked decrease in allergy in lesions of long standing. He also observed that in the occasional far advanced case of tuberculosis the animal did not react to tuberculin. He said, "The imperfections of tuberculin mentioned here are eclipsed by its good qualities. . . . Tuberculin, therefore, in spite of its imperfections, marks an immense advance in the diagnosis of tuberculosis. By its help we are able to discover a great number of cases of tuberculosis which were formerly absolutely concealed. Until something still better has been found, we must consider it to be our best weapon in the struggle against tuberculosis in cattle."

In this country the findings of Gutmann, Bang and others were first confirmed by Pearson and Cotton in Pennsylvania, 1892, and Russell in

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Wisconsin, 1893. Following these early demonstrations, the tuberculin test was widely used by veterinarians throughout the United States with overwhelming proof of its diagnostic value. At post mortem, lesions were found with such frequency in tuberculin reactors that a reaction became synonymous with the presence of lesions. However, just as Bang had observed, in the occasional reactor no lesion was found. At first, such animals were designated *no-lesion reactors*, but this was later changed to *no-visible-lesion reactors*, and now to *no gross lesion found*, because many veterinarians believe that in such animals lesions are present but are too small or located in too unusual places to be detected on routine necropsy. In fact, in 1923 Calmette said, "Tuberculin has frequently been accused of having given a false indication because no tuberculous lesions could be found . . . It was proved, however, long ago that in these circumstances the organs had not been searched with sufficient care . . . Whenever a tuberculin reaction is positive, there exists somewhere a follicular lesion or at least a gland containing tubercle bacilli whose presence can be disclosed by experimental inoculation of the guinea pig." Schroeder in 1924 cited a number of instances in which tuberculin reactions among cattle were not explained until long and tedious post mortem examinations, not possible at slaughter houses, revealed tuberculous lesions in unusual and unexpected locations and in organs of the body rarely involved. In 1924 Kiernan pointed out that the situation with reference to no-lesion tuberculin reacting cattle is not as serious as some have been led to believe, because, when based on the total cattle tested, possible error was only 0.08 per cent.

The term *nonspecific tuberculin reaction* was introduced. This supposedly covers the no gross lesion found (no-visible-lesion) reactor thought to be sensitized by nonpathogenic acid-fast bacilli. However, this term is in considerable controversy in the minds of many workers who feel there has not been adequate documentation of cases to justify its use.

There are numerous well-qualified veterinarians who believe that the animal which reacts to tuberculin, but presents no demonstrable lesion at routine necropsy, has lesions somewhere which might be found if more detailed post mortem examinations were made. This belief recently was partially supported by Nassal who made special post mortem examinations on the carcasses of 1000 cattle which had reacted to tuberculin but at routine necropsy showed no lesion. Extensive pathological and bacteriological examinations were made including lung and intestinal lymph nodes. In 50.2 per cent of all cases it was possible to demonstrate tuberculous lesions, of these, 86.6 per cent by gross pathological examination and 13.4 per cent by culture or animal tests. Of the lesions noted, 86.5 per cent were found in the lungs, 12.1 per cent in the intestinal lymph nodes and 1.4 per cent in the udder and udder lymph nodes. In 49.8 per cent of all cases the cause of the positive tuberculin reaction and the negative post-mortem results was not determined.

It is known that tubercle bacilli which usually do not produce progressive disease in a given species may cause slight nodulation and consequent tuberculoprotein sensitivity. For example, the human and avian types may cause this phenomenon in cattle. There is also some evidence that soil acid-fast bacilli may result in a slight degree of sensitivity.

One wonders if still more extensive and intensive examinations, particularly of unusual sites of the carcasses studied by Nassal, would have revealed lesions in the remainder of these carcasses and if it might have been possible to identify the type of acid-fast bacilli. Until such a study is done, the safe course seems to be to regard all tuberculin reactions as significant.

Between 1908 and 1912, Ghon did meticulous necropsies on the bodies of 184 children who had reacted to tuberculin but had no other evidence of the disease during life. He found lesions in 183; the remaining body was not completely examined. Ghon said, "From the standpoint of the pathological anatomist, my researches which in regard to the question are not limited to the cases described here, justify me in concurring entirely with those who advocate the specific value of the tuberculin reaction." The meaning of the tuberculin reaction in people is the same as that in cattle.

Preparation of Tuberculin

Koch prepared the tuberculin he announced in 1890 by growing tubercle bacilli in a broth medium made from the flesh of veal to which he added 0.5 per cent common salt, 1 per cent dried peptone, and 5 per cent glycerine. This medium was kept at a temperature of 98.6° to 102.2° Fahrenheit, and from 6 to 10 weeks the tubercle bacilli were allowed to multiply. He then heated the container and its contents to such a high degree that tubercle bacilli could not survive. The medium was next strained through ordinary filter paper and slowly evaporated to one-tenth of its original volume. The remaining substance was passed through a porcelain filter, and the filtrate was tuberculin. Obviously the tuberculin Koch prepared contained no tubercle bacilli dead or alive as all had been killed by heat and filtered from the liquid.

In 1925, Dr. Marion Dorset, Chief, Biochemic Division of the United States Bureau of Animal Industry, distributed tuberculin that he made on synthetic medium. He said there was no question of the remarkable efficiency of the former tuberculin as produced by Koch since it had brought about the reduction of bovine tuberculosis in the United States to a mere fraction of its former prevalence. However, there had been relatively few instances of reactors in which it had not been possible to find lesions and others where on a single test tuberculous animals failed to react.

The synthetic medium Dorset employed contained no protein. One advantage was that it produced three or four times as much tuberculin as the previous one. Another was that tuberculin produced in this manner contained essentially only substances derived from tubercle bacilli. In 1933 it was accepted by the National Tuberculosis Association. Following 1934, all tuberculin for the testing of cattle was made on synthetic medium. This new tuberculin was thermostable as no difference in potency was found when it was heated in streaming steam for 24 hours or autoclaved under 15 pounds steam pressure for three hours.

Beginning in 1908 the Bureau of Animal Industry tested regularly for purity and potency various brands of tuberculin manufactured under government license and that produced by the Bureau and sold in the United States.

At one time it was thought necessary to have tuberculin prepared from different types of tubercle bacilli in order to test accurately for disease produced by the various types in animals and man. For example, tuberculin made from the bovine type was thought valuable only in testing for disease produced by this organism. However, this theory was abandoned when it was found that tuberculin made from either the human or the bovine type of tubercle bacilli is equally good in testing for disease caused by the other type. Inasmuch as the human type grows more luxuriantly on culture medium than the bovine type, practically all of the tuberculin used to test cattle in this country has been made from the human type.

The situation is different in tuberculosis caused by avian type of tubercle bacilli, since animals and persons infected with this organism may fail to react to tuberculin made from the mammalian type but react definitely to that made from the avian type of tubercle bacilli.

Inasmuch as original tuberculin (OT) contains all the chemical fractions of tubercle bacilli and only the protein fraction is the active principal in the tuberculin reaction, a method was devised for producing a testing substance containing only tuberculoprotein. This was produced by Long and Seibert in the early 1930's and is known as purified protein derivative (PPD).

Methods of Administration

Subcutaneous

Koch employed the *subcutaneous* method of administration which was then used for more than a decade in testing people and animals. Reactions were local, focal, and constitutional. Inasmuch as allergy was not then understood, the large doses of tuberculin administered to people often resulted in such severe reactions as to reactivate old lesions and cause already active ones to progress rapidly.

Ophthalmic

In 1907 Wolff-Eisner and Calmette perfected the *ophthalmic* test which consisted of introducing concentrated tuberculin or a small compressed disc of precipitated tuberculin between the lids of one eye so it entered the conjunctival sac. The reaction was manifested by inflammation, lacrimation, and swelling of the mucous membrane followed by mucopurulent or purulent discharge.

Percutaneous

In 1908 Moro rubbed into the cleansed skin over an area of about 5 centimeters in diameter a pomade consisting of equal parts of tuberculin and lanolin. No dressing was applied. When a reaction occurred, it consisted of numerous small red papules which appeared in about 24 hours and persisted for several days. This is known as the *percutaneous* test. Lovet, 1929, reported a thoroughgoing study of the percutaneous method and concluded that when properly administered it is as satisfactory, or even more so, as the epidermal method described by Pirquet.

In 1908 Lautier bandaged to the surface of the skin a pledget of absorbent cotton moistened with a 1 per cent solution of tuberculin. The reaction is similar to that seen in Moro's method, but covers a much

smaller area. This method was revived by Vollmer and Goldburger under the name of the *patch* test in 1937.

In 1933 Wolff presented the *tape* test which consists of applying a particle of ointment about the size of a pea and containing tuberculin to thoroughly cleansed skin, then tightly applying adhesive tape about two inches square. Forty-eight hours later the tape is removed and a reaction is indicated by the presence of papules, erythema, and induration.

Numerous other ointments, jellies, etc., containing tuberculin have been used in the percutaneous method.

Epidermal and Intracutaneous

In 1907 Pirquet, Vienna, announced a procedure which consists of applying tuberculin to a superficial abrasion on the skin (*epidermal*). Infected persons presented only a local reaction within 24 to 48 hours usually consisting of induration and hyperemia at and around the site of application. Stewart introduced the *puncture* method by placing a drop of tuberculin on the skin and making several superficial needle punctures through it, thus permitting tuberculin to enter the layers of the skin. He found this to be a good testing procedure. This method has been revived by Heaf and an instrument is available for making multiple skin punctures of known depth in one operation.

Mantoux, 1908, presented the *intracutaneous* (intradermal) method of administering tuberculin for testing people. At the same time Moussu used this method for testing cattle. The reaction consists of induration or edema or both at the site of administration often surrounded by hyperemia.

Various other methods have been devised such as instilling tuberculin into the nose, the urethra, the vagina, and the rectum, but none has been found practical.

Evaluation of Different Methods of Administration

In testing cattle, the subcutaneous method continued in general use for a good many years. However, in 1914 the intracutaneous method became the official test in Montana. In 1920 the Bureau of Animal Industry and the United States Livestock Sanitary Association adopted this method after which subcutaneous administration was practically abandoned.

The ophthalmic test was continued for a while in conjunction with the intradermal test. However, by 1932 the Bureau of Animal Industry discontinued producing discs for this test. Since 1920 the Bureau, now Animal Disease Eradication Division, United States Department of Agriculture, has used original tuberculin (OT) made on synthetic medium and administered by the intracutaneous method. This, as a tubercle bacillus finder, has enabled veterinarians of this country to make an accomplishment that has been designated "man's greatest victory over tuberculosis."

Among people, the *subcutaneous* method of administration fell into disrepute after it was shown to have a deleterious effect on some tuberculous lesions. It was largely replaced by Pirquet's epidermal method which, for a good many years, was the main testing procedure. Then the

intracutaneous method of Mantoux won popularity and has since been considered the most accurate method of administering tuberculin, largely because of its measured dosage.

The intracutaneous method has usually consisted of administration of 0.1 of a cubic centimeter of a dilution of one part tuberculin in 999 parts of diluent which equals 0.1 milligrams of tuberculin. If no reaction is present within 72 to 96 hours, the second dose is administered consisting of 0.1 of a cubic centimeter of a solution consisting of one part tuberculin in 99 parts of diluent. This contains 1.0 milligrams of tuberculin. Smaller initial doses have been employed, as well as larger, than second doses by some workers. For example, persons who harbor unjustified fear of tuberculin administration may employ 0.01 or 0.001 of a milligram as the initial dose, whereas those who know that sensitivity may wane in long-standing infection may follow the usual second dose of 1 milligram with 2 or more milligrams.

Some workers prefer to express the dose of tuberculin in international units. For example, .0001 ml. of purified protein derivative equals 5 units (5 TU). When original tuberculin (OT) is used, the dose consists of 0.1 ml. of a 1-2000 dilution. One of the former criticisms of original tuberculin (OT) was that commercial preparations varied in potency. It is now known that this may also be true of PPD.

Since 1937 the patch test has been used quite extensively because of its ease of administration, etc. Usually it has not been found as accurate as the two-test dose administered by the intracutaneous method. However, workers have reported it to be as effective as the first intracutaneous test dose. There is good evidence that the patch test detects most persons who have a high degree of sensitivity such as infected school children and young adults whose sensitivity is usually at a high level. This also applies to other adults who have recently been initially infected.

As sensitivity is developing, a slight reaction to tuberculin may be obtained; whereas a much higher degree of sensitivity is elicited if the test is administered days or weeks later. Slight reactions may also be observed in persons suffering from acute forms of tuberculosis such as meningitis and miliary disease or those with chronic far advanced disease. In fact, under these conditions sensitivity may wane to such a degree that the usual second dose of OT or PPD is not adequate. Sensitivity also wanes when lesions of long standing exist and no reinfection has recently occurred. This is often seen in elderly persons even to the point of requiring more than 1 milligram of tuberculin to detect its presence. There is some evidence that sensitivity is depressed during attacks of acute infections. However, under none of the above circumstances is allergy completely lost. Indeed, it may disappear so completely that the tissues do not respond to 10 milligrams which strongly suggests that in such individuals tubercle bacilli have disappeared. Recent observations suggest that this occurs in more persons and in a shorter period of time than had previously been suspected.

The degree of sensitivity varies in the same individual depending upon such factors as recentness of infection, duration of infection, and conditions which temporarily depress it. Therefore, when one administers

the test to a large group of persons, one must expect to observe various degrees of sensitivity among those harboring tubercle bacilli.

More than a third of a century ago, it was well known that tuberculin, administered by the epidermal or intracutaneous method, separates those who are infected with tubercle bacilli from those who are not. Nothing or no one has disproved the findings established by testing and post mortem examination of reactors. Even in the last century, it was demonstrated that in animals the size of the tuberculin reaction is no indication of the extent or activity of the disease. No one has yet shown that the degree of reaction bears any relation to extent or progressiveness of tuberculous lesions in man, despite numerous efforts to make it do so.

Any hope that one might entertain of determining which tuberculin reactor, regardless of degree of reaction, already has or will later develop clinical disease is forlorn. Therefore the tuberculin reaction, again regardless of degree, is not the criterion as to which persons among the group of reactors should be examined periodically. All reactors must have this examination if one desires to find those in whom clinical disease is destined to develop while it is in the silent stage and before it has become contagious and when it is so treatable.

One sometimes wonders if the hardest and most confusing way of administering and reading tuberculin tests has been found. Already it has been so complicated as to partially lose its usefulness by discouraging physicians from employing it. After listening to a panel discussion in which meticulous and unnecessary detail was emphasized, one physician was heard to say that all of the testing of the past was worthless because of improper dosage, etc., which had been employed. How ridiculous, when everyone who has had long and extensive experience with tuberculin knows that it is and has long been our sheet anchor. Medical literature, both human and animal, abounds with large well-documented reports that testify to the value of the test, regardless of the method of administration, for more than 65 years.

For decades there have been controversies over methods of administering tuberculin, the dosage, how to interpret, etc., etc. Most of the controversy has been "Much ado about nothing." It has settled no important question, but has consumed much time that could have been better spent in administration of the test and acting accordingly.

With all the methods that have been employed for administration of tuberculin, including various dosages, the fact remains that it is the protein fraction of the tubercle bacillus to which tissues become sensitized and it is this fraction that is responsible for the reaction. Each method of administration, including variations in dosage, has had its proponents and opponents. For the person or animal highly sensitive to tuberculoprotein, it is only necessary to get tuberculin into the pores of the unabraded skin, into a superficial scarification, or within the layers of the skin.

When situations and circumstances make one method of testing impossible, but one slightly less effective is acceptable, enthusiasm for "perfection" should not be allowed to outrun judgment. Common sense should dictate that it is far better to conduct a program slightly below par than to lose all.

A review of some of the outstanding values of the tuberculin test strongly supports the program you adopted and pursued.

1. It detects the presence of tuberculosis after it appears in the human body earlier than any and all other phases of examination. It is an important event in a person's life to become a tuberculin reactor. If and when it occurs, it means that tuberculosis is already present in the individual's body. Knowing that tuberculous lesions are present, even though microscopic, places the physician on the alert and makes it mandatory that the reactor be examined periodically for the earliest possible evidence of clinical lesions if and when they occur.

2. Finding tuberculosis soon after allergy appears, enables the physician and the health officer to locate the source of the infection in adult associates much more often than by any other method. It is certain that the tuberculin reactor has been in contact, directly or indirectly, with a person who has contagious tuberculosis. This is important information, since the responsible person may be found by examining the reactor's contacts. If the reactor is an elderly person, the infection may have occurred in his childhood, and the responsible person may have been dead long since. However, the person of any age who has been tested periodically and converts to a tuberculin reactor since the last recent test has had the exposure during that time, and, in point of time, the contagious case is not far away. In the entire field of medicine, there is nothing more fascinating than picking up the trail of a case of tuberculosis by finding the tuberculin reaction and tracing it to its source.

3. The only way physicians and the courts can determine with accuracy the responsibility of infections that lead to litigation is through periodic tuberculin testing. The industry or institution that permits an employee, student, or other personnel member to be infected in line of duty is responsible for the clinical disease that may subsequently develop. Such infections are at first detectable only by the tuberculin reaction.

4. Periodic observation and reexaminations of persons who react to tuberculin enables the physician to find those destined to develop clinical lesions in the silent and noncontagious stage. This procedure solves the problem which so long has so harassed tuberculosis workers; namely, finding tuberculosis only after it reached an advanced stage.

5. The tuberculin test determines the magnitude of the tuberculosis problem in any group of people. It detects those who are harboring tubercle bacilli, and it is they who constitute the problem. Prior to your testing in the schools of St. Louis, no one ever knew the magnitude of the problem. It is now established with accuracy that about 5 per cent of the students have tuberculosis.

6. Periodic testing with tuberculin determines the effectiveness of a tuberculosis control program. Testing before the program starts fixes the base line. Subsequent testing determines whether the incidence of infection is decreasing or increasing from year to year. Your retesting of the nonreactor children in the St. Louis schools is of great value in providing such information.

7. Already many physicians are administering antituberculosis drugs for recent tuberculous infections when lesions are usually microscopic and

vascular. Obviously, this is the only time when all tubercle bacilli may be reached by drugs, and only the tuberculin test tells when recent infection has occurred. As yet, there is no evidence that present drugs are germicidal, but they may suppress tubercle bacilli enough to prevent the occasional acute reinfection clinical type of disease, such as meningitis.

These and many other values justify the statement that the tuberculin test is the master key which unlocks every door leading to the eradication of tuberculosis. Hence your program is built on solid rock.

St. Louis Academy Accomplishments

Today I learned that since your project began, your members, the St. Louis County Tuberculosis and Health Society and the St. Louis County Health Department, have cooperated in administering 200,000 tuberculin tests in the St. Louis city and county schools. This you have done as a *public service*. No member has charged anyone for his work. This is a fine demonstration of the true spirit of the medical profession which should be in operation everywhere.

You have found that about 5 per cent of the school children of St. Louis react to tuberculin and, therefore, have at least microscopic tuberculous lesions containing living tubercle bacilli. This means that in each 1000 students, you have found 50 cases of tuberculosis among some of whom clinical disease will develop before they complete their life span.

Persons who are harboring tubercle bacilli have been spoken of as carrying concealed weapons—weapons which are at all times dangerous to them and may at any time become dangerous to their associates. In the schools of St. Louis, you have found about 5 per cent of children carrying such weapons, and among their associates, you have often found the persons previously carrying concealed weapons which did them harm and which caused them to injure children. This train of events can be stopped in large part among the 5 per cent you have found.

By a well-established method of follow-up, 95 per cent of those destined to develop chronic pulmonary disease can have their lesions detected while they are silent, noncontagious and will respond promptly to treatment. This is in sharp contrast to the preceding generations of children for whom sanatoriums later had to be built, whose tuberculosis mortality reached such high levels, and whose present survivors, now elderly people, are still contributing most of the tuberculosis morbidity and mortality.

Wood and Mantz sought the sources of infection of kindergarten and first grade school children who reacted to tuberculin. They found 10 times more clinical cases among the adult associates of these little children than had ever been found by mass x-ray surveys. Katharine Hsu, Houston, Texas, reported that the yield of clinical cases of tuberculosis by x-ray survey of the general population was 0.09 per cent in 1955; whereas, among the members of families whose children had active primary tuberculosis, the yield was 25 per cent among the associates. In one of your earlier reports, it was stated that by seeking the sources of the infections in children among their adult associates and examining the older children who reacted to tuberculin, you found 10 times more clinical cases of

tuberculosis than were ever found in St. Louis mass x-ray surveys. This is an excellent by-product yield; however, the main return of any tuberculin testing program is the tubercle bacilli located in the bodies of the reactors. This group of five per 100, which you have already found, is in the budding stage of tuberculosis and can be prevented from producing ripe and over-ripe fruit.

Work of Physicians in General Practice Imperative

As sanatoriums developed and tuberculosis became a so-called medical specialty, an exceedingly unfortunate situation evolved. It was the feeling among the "experts" operating sanatoriums or working in their private offices and clinics that physicians in general practice had little or no ability or training in the diagnosis and treatment of tuberculosis. Even those who manifested a good deal of interest were suppressed. The physician in general practice often permanently lost all contact with his patient. He was so discouraged at every turn that despite his fine medical training, often greatly enhanced by experience, he himself came to believe that he probably was not capable and certainly was not wanted.

We had failed to take cognizance of Dr. Trudeau's statement, "On the general practitioner and the dispensary physician rests a great responsibility of detecting the disease in its incipiency. It is to them and not to the specialist that the patient first applies."

In a few areas, general practitioners have been allowed to retain their rightful position. A sanatorium district of 250,000 people may be cited where, for more than a third of a century, the medical director recognized the importance of general practitioners and acted accordingly. In this sizeable geographic area, the office of every physician was and is a tuberculosis center where the latest information on diagnosis, treatment, and prevention is known and practiced. There, the lowest mortality, morbidity, and infection attack rates exist in the state.

There are approximately 90,000 general practitioners in the United States. Almost overnight they could become a powerful force against the tubercle bacillus. Obviously the few chest specialists in the United States cannot solve the medical aspects of the problem alone. Participation of all other physicians, and especially general practitioners, is imperative.

Not enough emphasis has been placed upon tuberculosis work done in the offices of physicians and hospitals where more tuberculin testing, x-ray film inspections, and final diagnoses have been made than in surveys. Surveys have been of great value as demonstrations, but the best case finding of the future should continue to be practiced in the offices of physicians, hospitals, permanent clinics, and special projects such as yours.

It was a significant event in the tuberculosis eradication movement when the St. Louis Academy of General Practice entered this field. While several smaller groups of physicians in general practice have participated and demonstrated their extreme value, yours is the first large well-organized group attacking the tubercle bacilli of a large city and county in cooperation with the County Tuberculosis Society and Health Department.

Already you have made the finest demonstration of the capability of a large group of physicians in tuberculosis work of any city in this country.

Coasting on Laurels

It is true that there has been continuous decrease in tuberculosis mortality, morbidity and infection attack rates for the past several decades. However, the morbidity and mortality as well as infection attack rates decrease until about 1947 were largely the result of isolation and treatment of tuberculous patients in sanatoriums and the control of the disease among cattle. Both served as terrific foes of tubercle bacilli by tremendously reducing their population. Many were prevented from reaching the bodies of children to perpetuate themselves as they had done for centuries. Those found in the carcasses of millions of cattle were promptly destroyed, while those in the bodies of large numbers of people were corralled. Thus, the protected generations of children exhibited a marked decrease of tuberculous infection and a corresponding decrease of morbidity and mortality as they attained early adulthood and thereafter.

Up to about 1947 morbidity and mortality were governed by the incidence of tuberculous infection, and this is still true of morbidity. Beginning about 1947 the divergence between mortality and morbidity began to appear due to the sharp and almost precipitous decrease in mortality. Although not understood in the beginning, and sometimes erroneously credited to other factors, this decrease proved to be due to antituberculosis drugs; and, in some cases, to resectional surgery. These procedures had at least postponed death of many persons who otherwise would have died. It is too soon to know how many of these persons, at least temporarily spared, will later die from tuberculosis.

There would have been just as precipitous a decrease in morbidity if a drug or some other method had become available in 1947 that would prevent the development of clinical disease in infected persons. In the absence of such a factor, the decrease in morbidity continued as in the past few decades, and is likely to go on in proportion to the number of infected persons.

Thus, for some decades, we have been resting on our laurels, enjoying the small but continuous decrease of tuberculosis in all its phases resulting from methods initiated early in the 20th century.

A Forward Plunge Overdue

The time has been right for a plunge forward for more than a quarter of a century, and attention has frequently been called to a well-established procedure capable of striking another terrible blow to the populations of tubercle bacilli. The procedure consists of finding adult tuberculin reactors, examining them periodically and detecting lesions while they are without symptoms, noncontagious and easily controlled. This procedure was exceedingly effectively practiced in the preantituberculosis drug days and decades. It can now be carried out with considerable time saving with addition of drugs. As long as we continue to rest on our laurels and fail to take advantage of the above procedure and do little, if any, more work this year than we did last year, tuberculosis may be

expected to continue to slowly decrease until the effect of the work of the sanatorium and the veterinarian are spent, after which an upturn may be predicted.

To put an end to this lethargy, a program should be developed quickly and forcibly, which will be as effective in the future in reducing tuberculosis as the sanatorium movement and the control of disease among animals were in the past.

The stage has long been set for a "plunge forward" in the tuberculosis eradication movement. Except for the introduction of antituberculosis drugs and resectional surgery which are still on trial, there has been little stepping up of the drive against tuberculosis in the last few decades. Throughout this period, each community, county and state did about the same amount of work each year with the same program and with perhaps slightly increased funds, as was done the year before—barely scratching the surface of the tuberculosis problem. During this period all necessary information and tools were available to strike powerful blows and markedly reduce the populations of tubercle bacilli. Unfortunately, controversies were waged as to efficacy of well established procedures and, except in a few places, no one seemed to have the courage and initiative to proceed with markedly accelerated speed toward the eradication goal.

The attitude of occasional members of our own profession in many places prevented the launching and perpetuation of such a program. Even though the goal was eradication of a disease which has been one of the most serious afflictions of humanity since antiquity, some physicians demanded exorbitant sums for their part in relieving the world of this curse.

Fortunately not all physicians seek to profit in a program designed to solve a medical problem that threatens their nation's future. Many physicians, probably the majority, recognize that tuberculosis is a menace to them and their families to the same degree as to the remainder of the citizenry. They realize that they also have a unique part to contribute to such a program unavailable to others; namely, diagnosis. They are willing to give this service while persons engaged in other activities of life contribute all of the other work necessary to complete the program.

Eighteen years ago, the physicians of Meeker County, Minnesota, conducted a county-wide tuberculosis program, including persons of all ages, consisting of the tuberculin test, x-ray film inspection of the chests of reactors and completion of examinations as indicated. This was done as a public service. It paid excellent dividends, not only in locating the tubercle bacilli and the clinical cases, but also by winning the goodwill of the public and by increasing their office work subsequently through the confidence they established in the citizenry in their ability to diagnose and treat this disease. Physicians in other counties and municipalities have since contributed their services in tuberculosis eradication campaign demonstrations.

If your program is continued and can be extended to the entire citizenry of St. Louis, this city could soon complete a demonstration of tuberculosis control far superior to that of any city in America. You have created so

much interest in tuberculosis among the citizenry of St. Louis that your Tuberculosis and Health Association will find it easy going to develop an all-out program which will exceed anything accomplished elsewhere. Your work has been so valuable and the spirit manifested so laudable, that as the years pass your organization probably will always be looked upon as a pioneer in this activity which ultimately should be adopted by all local and state groups, as well as the National Academy of General Practice. Just as in St. Louis, this could become the strongest offensive attack on tuberculosis among people of all time anywhere on earth.

Certification of Schools

A project that is rapidly gaining popularity could be of inestimable value in St. Louis. Working in close cooperation, the American School Health Association and State and Municipal Tuberculosis and Health Associations have developed a project which consists of administering the tuberculin test to at least 95 per cent of the school children and 100 per cent of the personnel. When this is accomplished, schools receive official Class A Certificates. Those schools which for one reason or another cannot meet these qualifications may be given certificates of lower grade. Certification is not based upon the amount of clinical tuberculosis found or the percentage of tuberculin reactors but upon the control program in progress. These official certificates are now displayed on the walls of several thousand schools. The program has proved superior from the standpoint of insuring completeness of response of children and personnel to anything leading to the eradication of tuberculosis that has ever been done in any community.

Completeness of respnose is extremely significant in any program, otherwise the most dangerous cases may be missed. The most important group in any school is the personnel since silent or even extensive pulmonary tuberculosis is far more likely to be present in this group than in children. Therefore the certification project requires inclusion of every personnel member.

With a third of our citizens harboring tubercle bacilli, the solution of the tuberculosis problem is a part of our national defense. Even though there has been marked reduction in mortality, morbidity and infection attack rates, the annual loss from the disease is still stupendous and will continue so until the medical profession everywhere adopts an adequate program for people of all ages and carries it through to eradication. The importance of the physician in general practice is not a new thought. It was expressed by Osler as follows:

> "A last word on the subject of tuberculosis to the general practitioner. *The leadership of the battle against this scourge is in your hands. Much has been done, much remains to do. By early diagnosis and prompt, systematic treatment of individual cases, by striving in every possible way to improve the social condition of the poor, by joining actively in the work of the local and national antituberculosis societies you can help in the most important and the most hopeful campaign ever undertaken by the profession.*" This statement is as true today as when it was written.

SUMMARY

1. Soon after Koch introduced tuberculin in 1890, it was extensively used in testing animals in several nations. Necropsy of both reactors and nonreactors revealed that it was highly specific for tuberculosis but the degree of reaction had no constant relationship to extent of disease. These observations have since been confirmed by test and necropsy of many million animals. From 1917 to 1957 inclusive, veterinarians of the United States alone administered 387,803,473 tuberculin tests. The carcasses of the 4,062,634 reactors were examined. Ghon, 1912, Calmette, 1923, and many others have emphasized the values of the tuberculin test in both people and animals.

2. Meticulous necropies have revealed lesions in many human and animal reactors which were missed on routine post mortem examinations thus making the use of such terms as no lesion and nonspecific reactors more questionable.

3. Original tuberculin (OT) and purified protein derivative (PPD) have been used extensively since 1890 and the early 1930's respectively. There is no significant difference between them in reactions elicited when properly prepared in ability to produce reactions.

4. Methods of administration have included subcutaneous, ophthalmic, percutaneous, epidermal, and intracutaneous. The percutaneous procedure now in most common use is known as the patch test. The intracutaneous is now the most popular method. The tuberculin reaction, regardless of the method employed, has stood the test of time. It has been and still is the master key for eradication of tuberculosis.

5. The entry of the St. Louis Academy of General Practice into the schools in cooperation with the County Health Department and Tuberculosis Society is an historic event in the world-wide tuberculosis field. Your work has been done as a public service for your city, state, and nation. The demonstration made here constitutes an example which should be followed by physicians in general practice everywhere.

RESUMEN

1. Después de que Koch en 1890 introdujo la tuberculina, bien pronto se usó ampliamente para las pruebas en animales en varios países. La necropsia de los reactores y de los negativos, reveló la gran especificidad de ella en la tuberculosis pero el grado de la reacción no estaba en relación con la magnitud de la enfermedad.

Estas observaciones se han confirmado por la reacción y por la necropsia en muchos millones de animales. De 1917 a 1957 inclusive, los veterinarios de los Estados Unidos tan sólo hicieron 387.803.473 reacciones de tuberculina. Se examinaron los cuerpos de los animales que reaccionaron en número de 4.062.634.

Ghon en 1912, Calmette en 1923 y muchos otros han recalcado el valor de la reacción tuberculínica en las personas y en los animales.

2. Las necropsias meticulosas han revelado en muchos hombres y en animales reactores que tenían lesiones que habían sido pasadas por alto en la necropsia rutinaria, haciendo así que tales términos como la ausencia

de lesión o la posibilidad de reactores no específicos fuesen más objetables.

3. La tuberculina original antigua (OT) el derivado purificado proteínico (PPD), se han usado ampliamente desde 1890 y principio de 1930, respectivamente.

No hay diferencia significante entre ellas en las reacciones que provocan cuando se preparan convenientemente.

4. Los métodos de administración han incluído el subcutáneo, oftálmico, percutáneo, epidérmico e intracutáneo. El procedimiento percutáneo que más se usa ahora, es el llamado de la reacción del parche. El intracutáneo es el más poular ahora. La reacción tuberculínica cualquiera que sea el método que se use, ha resistido la prueba del tiempo. Ha sido y aún es, la llave maestra para la erradicación de la tuberculosis.

5. La entrada de la Academia de Práctica General de San Luis, en las escuelas constituye un evento histórico en el campo mundial de la tuberculosis. Vuestro trabajo se ha hecho como un servicio público a vuestra ciudad, vuestro estado y a la nación. La demostración hecha aquí es un ejemplo que debe seguirse por los médicos en la práctica general en todas partes.

RESUME

1. Aussitôt après que Koch eût découvert la tuberculine en 1890, elle fut utilisée en grand pour rechercher les réactions des animaux dans plusieurs pays. L'autopsie tant de ceux qui avaient des réactions positives que de ceux dont les réactions étaient négatives révéla qu'elle était hautement spécifique de la tuberculose. Toutefois le degré de réaction n'était pas en rapport constant avec l'étendue de la maladie. Ces observations ont été confirmées depuis par les tests et les autopsies de plusieurs millions d'animaux. De 1917 à 1957 inclus, les vétérinaires des Etats-Unis ont pratiqué 387.803.473 tests tuberculiniques. 4.062.634 animaux allergiques furent examinés. Ghon en 1912, Calmette en 1923 et beaucoup d'autres ont mis l'accent sur la valeur du test tuberculinique à la fois chez l'homme et chez l'animal.

2. Des autopsies méticuleuses ont révélé des lésions chez beaucoup d'hommes et d'animaux réagissant à la tuberculine, qui avaient passées inaperçues lors des examens post-mortem de routine. C'est pourquoi il nous parait discutable d'utiliser les termes "d'individus allergiques sans lésions," ou "d'allergiques non spécifiques."

3. La tuberculine originale (OT) et son dérivé protéinique purifié (PPD) ont été utilisés extensivement l'une depuis 1890 et l'autre dès le début de 1930. Il n'y a pas de différence importante entre elles dans les réactions déterminées quand elles sont convenablement préparées dans le dessein de créer de telles réactions.

4. Les méthodes d'administration ont compris les voies souscutanées, ophthalmiques, percutanées, épidermiques et intracutanées. Le procédé percutané d'emploi le plus fréquent maintenant est connu sous le nom de "patch test." La voie intracutanée est actuellement la méthode la plus populaire. La réaction tuberculinique, sans égard à la méthode utilisée, a subi l'épreuve du temps. Elle a été et est encore la clé de l'éradication de la tuberculose.

5. L'entrée de l'Académie St-Louis de Médecine Générale dans les écoles est un évènement historique dans le domaine mondial de la tuberculose. Votre travail a été celui d'un service public pour votre ville, votre état et votre nation. La démonstration faite ici constitue un exemple qui devrait être suivi partout par les praticiens de médecine générale.

ZUSAMMENFASSUNG

1. Bald nachdem Koch das Tuberkulin im Jahre 1890 eingeführt hatte, benutzte man es in verschiedenen Nationen intensiv, un Tiere zu testen. Autopsien sowohl von Fällen mit positiver wie mit negativer Reaktion ergaben, dass das Tuberkulin in hohem Masse für Tuberkulose spezifisch ist, jedoch steht die Stärke der Reaktion in keiner konstanten Beziehung zum Ausmass der Erkrankung. Diese Beobachtungen wurden seitdem bestätigt durch Teste und Autopsien von vielen Millionen Tieren. In der Zeit von 1917 bis einschliesslich 1957 führten allein die Tierärzte der Vereinigten Staaten 387 803 473 Tuberkulinproben aus. Die Kadaver von den 4 062 634 Tieren mit positiver Reaktion wurden untersucht. Ghon, 1912, Calmette, 1923 und viele andere haben den Wert der Tuberkulinprobe für Mensch und Tier hervorgehoben.

2. Sehr genaue Autopsien bie vielen Menschen und Tieren mit positiver Reaktion haben zur Entdeckung von Herden geführt, die bei routinemässiger Untersuchung post mortem übersehen waren, und stellten den Gebrauch solcher Bezeichnungen wie "ohne Befund" und "unspezifische Reaktoren" mehr in Frage.

3. Das Originaltuberkulin (OT) und das gereinigte Proteinderivat (PPD) wurden seit dem Jahre 1890 bzw. Anfang der 1930iger Jahre intensiv verwandt. Es besteht zwischen beiden kein wesentlicher Unterschied hinsichtlich der ausgelösten Reaktion bei ordnungsgemässer Herstellung betr. die Fähigkeit zur Bewirkung von Reaktionen.

4. Die Methoden der Anwendung umfassen die subkutane, okulären, perkutanen, epidermalen und intrakutanen Wege. Die perkutane Methode, die jetzt am meisten in Gebrauch ist, ist bekannt als Pflasterprobe. Die intrakutane Methode ist jetzt am meisten populär. Die Tuberkulin-Reaktion, unbeschadet der angewandten Technik, hat die zeitliche Bewährungsprobe bestanden. Sie war und ist noch der Hauptschlüssel zur Ausmerzung der Tuberkulose.

5. Die Aufnahme der Akademie für Allgemeinpraxis von St. Louis in die Schulen bedeutet ein historisches Ereignis auf dem weltweiten Gebiet der Tuberkulose. Ihre Tätigkeit geschah als öffentlicher Dienst für ihre Stadt, das Land und die Nation. Die hier erfolgte Kundgebung stellt ein Beispiel dar, das von den Ärzten in der freien Praxis überall befolgt werden sollte.

Cardiorespiratory Studies in Hamman-Rich Syndrome*

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In 1944, Hamman and Rich[1] reported four cases which they labeled as "diffuse interstitial fibrosis of unknown etiology." While our knowledge of the etiology of this entity has since advanced no further, our understanding of the syndrome has been enriched by the subsequent report of additional cases.[2, 3, 4] The clinical picture of progressive cardio-respiratory failure, and the pathologic picture of diffuse interstitial fibrosis are well-established. Our interest here, however, is to delineate the *physiologic* consequences of this disease, as they are demonstrated by detailed cardio-pulmonary function studies in two patients.

Hamman and Rich, in their original report, noted that these patients had desaturation of the arterial blood at rest. Austrian, McClement, Renzetti, Donald, Riley and Cournand[5] then indicated that "diffusion insufficiency for oxygen" occurred in a number of conditions characterized by diffuse pulmonary fibrosis, and they suggested that such a disorder might exist in Hamman-Rich disease. Subsequently, several instances of this disease have been reported in which hemodynamic and ventilatory studies were performed.[6, 7, 8] However, these reports have not sought to define the relationship between the alveolo-respiratory abnormalities and the hemodynamic aberrations, though dysfunction in both spheres was noted to co-exist. Combination of cardiac and pulmonary study in the individuals presented here has led to certain basic conceptions regarding the physiologic significance of diffuse interstitial fibrosis.

Case 1: C. D., a 52 year old white man, was found to have mild clubbing of the fingers on a routine examination. Close questioning elicited a history of insidiously progressive easy fatiguability over the preceding five years. Symptoms were not present in his daily routine as a business executive, but any form of exercise, such as playing golf, led to mild dyspnea.

Aside from the mild acral clubbing and minimal cyanosis evoked by performance of an exercise test, physical examination was within normal limtis. A chest roentgenogram demonstrated a diffuse reticulation throughout both lung fields (Figure 1A). The cardiac shadow was normal. An electrocardiogram demonstrated no abnormality of rate, rhythm or axis. Routine laboratory studies were unremarkable. Skin tests and other special search for an etiologic agent were unrewarding. A lung biopsy was therefore obtained. Microscopic study of the specimen revealed a diffuse interstitial infiltration with small intervening areas of normal pulmonary parenchyma. The infiltration was predominantly lymphocytic, but contained some plasma cells, eosinophils and a few neutrophils. Small zones of fibrosis were present within the infiltrated regions. The alveolar walls showed cuboidal lining, frequently, with some being covered by a hyaline-like membrane. There was edema of the interstitium. Many capillaries were either obstructed or thrombosed, while others were engorged and dilated. These alterations were consistent with a diffuse interstitial pneumonitis and fibrosis of the Hamman-Rich type.

A complete cardio-pulmonary function evaluation was carried out on October 9, 1956, the results of which are listed in Table I.

*Presented at the 23rd Annual Meeting, American College of Chest Physicians, New York City, May 29-June 2, 1957.

From the Cardiopulmonary Laboratory of the Pulmonary Disease Division, District of Columbia General Hospital and the Departments of Medicine, Georgetown University School of Medicine and George Washington University School of Medicine, Washington, D. C.

TABLE I

G. D. 52 y. o. W M weight 70 Kg. Body surface 1.8 m^2
height 170 cm.

RESULTS OF COMBINED CARDIOPULMONARY FUNCTION STUDIES ON PATIENT G. D., BEFORE THERAPY AND AFTER APPROXIMATELY SIX MONTHS OF STEROID TREATMENT

	October 9, 1956			March 29, 1957			
	Rest Room Air	Rest High O_2	Room Air Exercise	Rest Room Air	Rest 14.17% O_2	Rest High O_2	Exercise Room Air
Brachial Artery							
O_2 saturation per cent	92.5	99.5	89.9	97.3	88.3	100.0	91.7
pO_2 mm. Hg.	87		80	92	50		58
pCO_2 mm.Hg.	33.8	38.2	33.4	34.0	33.0	35.0	35.7
pH	7.45	7.42	7.44	7.45	7.45	7.40	7.40
Alveolo-respiratory Function							
O_2 uptake cc./min. STPD	265		905	219	195		744
Resp. Quotient	.72		.96	.84	.90		1.05
Minute Ventilation cc. BTPS	8330		35000	10920	9045		32475
Alveolar Ventilation cc./min.	4850		22200	4670	4570		18830
Alveolar p02 mm. Hg.	106	over 400	116	112	66	over 400	116
Alveolo-arterial p02 gradient mm. Hg.	19		36	20	16		58
Hemodynamic Function							
A-V Diff.02 Vol. per cent	4.85		10.38	5.0	5.17	4.15	5.97
Pulm. Blood flow L/min.	5.48		8.72	4.4	3.8		12.4
Pressure "Wedge" mm. Hg.	4.5		7.6	8.5	8	8	8
Pulm. Artery mm. Hg.	25/13		53/13	26/9	37/14	24/8	63/17
	15.4		26.3	17	23	15	34.5
Right ventricle mm. Hg.	26/—2		53/—5	26/—4			65/2
Right auricle mm. Hg.	—3		—3	0			0
Resistance, total lung dynes. sec. $cm.^{-5}$	225		241	310	485		220
Resistance, arteriolar	160		171	155	315		170
Right ventricular work Kg.m./min.	1.35		3.45	1.0	1.2		5.85
Heart rate	65		102	90	85	90	125

In view of the clinical improvement obtained by others with steroid therapy in similar cases,[2, 3] he was started and has been maintained on prednisone in a dosage of 20 mgm. per day. He experienced considerable subjective improvement on this regimen, stating that he no longer felt fatigued and experienced no dyspnea on moderate exertion. Chest roentgenograms demonstrated some clearing of the pulmonary infiltration (Figure 1B).

After six months of such therapy, combined cardio-pulmonary function testing was again carried out. The findings at this time are detailed in Table I.

Case 2: J. B., a 63 year old white woman, had first noted the onset of dyspnea on exertion some three years prior to our examination. Since that time, dyspnea, accompanied by weakness and anorexia, had been steadily progressive. She also complained of persistent, hacking, non-productive cough. A chest roentgenogram at the onset of these symptoms had demonstrated a diffuse, reticular pulmonary lesion. Six months prior to admission, a tentative diagnosis of Hamman-Rich Syndrome had been made. Subsequently, two trials of steroid therapy had led to moderate symptomatic improvement, with rapid deterioration when steroids were discontinued. She was first seen following withdrawal of such therapy for the second time.

Physical examination revealed a cachectic white woman with marked dyspnea, cyanosis and clubbing of the fingers. On cardiac auscultation, the second pulmonic sound was found to be markedly accentuated, and a ventricular diastolic gallop was heard. No murmur was present. Throughout the lungs dry inspiratory and expiratory rales were audible.

An electrocardiogram demonstrated a sinus tachycardia and right bundle branch block. Complete blood count and urinalysis were within normal limits. A chest roentgenogram showed a diffuse reticular infiltration throughout both lung fields with small cystic areas visible at the periphery (Figure 2). Skin tests and other studies directed toward establishing an etiologic factor were unrewarding.

She was again started on steroid therapy, a dosage of 20 mgm. of prednisone per day being employed. Combined cardio-pulmonary function studies were performed, the results being listed in Table II. A lung biopsy had been contemplated, but on the basis of these studies was rejected as too hazardous.

Her subsequent course was one of progressive pulmonary insufficiency and right heart failure. Death intervened 60 days after admission, making the total duration of the illness some three and one half years.

Autopsy: At autopsy, the pertinent findings were limited to the lungs and heart. The lungs were greyish in color and covered with multiple small cysts. They did not collapse when the chest was opened, and were quite rubbery in consistency to palpa-

FIGURE 1A

FIGURE 1B

Figure 1A: Close-up photograph of chest roentgenogram before therapy, demonstrating diffuse reticular infiltrate which was present throughout both lung fields.—*Figure 1B:* Repeat roentgenogram after approximately six months of steroid therapy demonstrating clearing of the infiltration.

tion. On gross section, there was an impression of diffuse fibrosis with some foci of emphysematous cystic change. The heart showed marked right ventricular dilatation and hypertrophy.

Microscopic examination of the lung demonstrated diffuse interstitial fibrosis with minimal evidence of interstitial round cell infiltration. There was a paucity of blood vessels, with those which remained being dilated and engorged. Superimposed on these alterations there was evidence of acute bronchopneumonia.

Discussion:

These two cases thus represent extremes of the Hamman-Rich spectrum. One was studied at "end-stage," shortly before death. The other had cardio-pulmonary evaluation at a time when he was virtually asymptomatic.

As can be seen in table I, the first patient had normal alveolar ventilation, as attested by the normal alveolar gas tensions. He achieved an arte-

TABLE II

J. B. 63 y.o. W/F weight 39 Kg. Body surface 1.3 m^2
height 150 cm.

RESULTS OF CARDIOPULMONARY FUNCTION STUDIES IN PATIENT J. B.

	December 13, 1955		
	Rest Room Air	Rest High O_2	Rest after Hexamethonium
Brachial Artery			
O_2 saturation per cent	74.7	100	80.2
pO_2 mm. Hg.	40		47
pCO_2 mm. Hg.	35.5	36.9	35.4
pH	7.46	7.44	7.45
Alveolo-respiratory function			
O_2 uptake cc./min. STPD	140		141
Resp. Quotient	.84		.82
Minute Ventilation cc. BTPS	14300		15100
Alveolar Ventilation cc./mm.	2840		2830
Deadspace/Min. Ventilation	.732		.815
Alveolar pO_2 mm. Hg.	110		109
Alveolo-arterial pO_2 grad. mm. Hg.	70		62
Hemodynamic function			
A-V Diff. O_2 Vol. per cent	7.27	7.30	8.79
Pulm. Blood Flow L/min.	1.9		1.6
Pressure "wedge" mm. Hg.	7		6
Pulm. Artery mm. Hg.	63/22	62/22	42/22
Right Ventr. mm. Hg.	63/0		41/0
Right Auricle mm. Hg.	2		2
Resistance total dyn. sec. $cm.^{-5}$	1450		1400
arteriolar	1160		1100
Right Ventr. Work kg. m. min.	.68		.57
Heart Rate	89		80

rial 02 saturation of 99.5% with high oxygen breathing. This suggests, that the slightly decreased resting arterial saturation and the resting A-a gradient cannot be adequately explained by veno-arterial shunting. This thesis is supported by the arterial 02 changes during exercise. At rest, normal values for pulmonary artery pressure and pulmonary vascular resistance were present. The gradient between alveolar and arterial oxygen tensions was also normal. When this patient is studied during exercise, one sees that the pulmonary bed is called upon to accept an increased pulmonary blood flow. It responds abnormally. The pulmonary arterial pressure rises to abnormally high levels, though the pulmonary wedge pressure remains normal, thus demonstrating a pre-capillary form of pulmonary hypertension. Furthermore, the calculated resistance between pulmonary artery and wedge remains fixed, rather than exhibiting a normal *decrease*. Simultaneously, pulmonary function studies demonstrated that the patient had developed arterial desaturation and pathologic widening of the alveolo-arterial oxygen tension gradient. This widening of the gradient reflects a lack of *equilibration* between alveolar and capillary oxygen tensions.

From such data, one must conclude that the disease process has severely limited the capacity of the pulmonary vascular bed. When challenged by an increased pulmonary blood flow, the bed is incapable of further distension, for its resistance remains fixed. It can accommodate the greater flow only by an increase of pressure. Such an adjustment requires that the velocity of blood flow be accelerated. This acceleration is reflected in a widening of the alveolo-arterial oxygen gradient, indicating that flow is so rapid as to deny adequate contact time between capillary blood and

FIGURE 2: X-ray of the chest of patient J. B. demonstrating diffuse pulmonary fibrosis.

alveolar gas. In the face of diminished contact time, complete oxygen equilibration cannot take place between alveolus and capillary blood. Thus, it is indicated that reduction of capillary bed in this individual has led to a fixation of pulmonary resistance. In this fixed resistance system, equilibration or contact time permitted becomes a function of the *pulmonary blood flow* which the reduced capillary bed is called upon to accept.

It is instructive to note that, despite the subjective improvement which this patient experienced, there has been no amelioration of his cardiopulmonary dysfunction. The "arteriolar" resistance values have remained identical in the second study. Considering the differences in cardiac output, the degree of diffusion insufficiency, as evidenced by the widened A-a gradient, is unaltered. It is plain that no functional improvement has accrued from steroid therapy, though one might propose that such treatment has prevented further progression of the process during the six month period.

Our second case demonstrates more advanced impairment. Alveolar ventilation is actually increased, causing a mild respiratory alkalosis. A veno-arterial shunt is again excluded by high oxygen breathing. Yet, despite normal ventilation, profound abnormalities exist even at rest. The alveolo-arterial oxygen gradient is abnormally wide, and is reflected in marked arterial desaturation. Pulmonary arterial hypertension is present and resistance is high. As this patient was unable to exercise, she was studied in a manner opposite to the first patient. That is, her pulmonary blood flow was lowered. This decrease in blood flow was accomplished by infusion of the ganglionic blocking agent, hexamethonium. When pulmonary blood flow was diminished, the pulmonary vascular resistance remained fixed at high levels. However, the pulmonary arterial pressure fell as blood flow reduced. Simultaneously, the alveolo-arterial oxygen gradient narrowed. Right ventricular work, being a function of pressure and flow, fell exponentially. Again, the dynamics of these changes are apparent. In a fixed-resistance pulmonary bed, such as this patient has even at rest, a lowering of pulmonary blood flow requires a *smaller* pressure gradient between pulmonary artery and capillary. At the same time, the velocity of blood flow must diminish. This allows a longer period of contact time. Alveolar and arterial oxygen tensions equilibrate more completely, and the A-a gradient narrows.

These functional studies in two patients at different points on the Hamman-Rich continuum demonstrate clearly the interrelationship of diffusion insufficiency for oxygen and pulmonary hemodynamics. They indicate that the predominant factor in the pathogenesis of these functional abnormalities is a decrease in the total cross-sectional area of the vascular bed, rather than a thickening of the alveolo-capillary interphase. It is the relationship between this restriction of vascular bed and pulmonary blood flow which determines both the cardiac and pulmonary aberrations, including the alterations in alveolo-arterial oxygen gradient. It is to be recognized that the state of the alveolo-capillary interphase remains unaltered during these studies. The basic anatomic disorder is not, therefore, one of alveolar thickening causing a true *block* of oxygen diffusion, but rather one of marked capillary loss leading to fixation of pulmonary resistance. The physiologic consequences of this anatomic compromise

can be heightened or reduced by elevating or decreasing the blood flow which the pulmonary bed is called upon to accommodate.

These findings emphasize the importance of simultaneous evaluation of alveolo-respiratory and hemodynamic function. Furthermore, they indicate that the most rational therapeutic approach in these individuals involves: (1) attempts to improve the anatomic defect; and (2) failing this, the reduction of pulmonary blood flow, through restriction of activity or with pharmacologic agents such as hexamethonium. Only by lowering pulmonary blood flow can one improve diffusion insufficiency and hope to avoid the relentless progression of cor pulmonale due to increased right ventricular work.

SUMMARY

Two cases of Hamman-Rich Syndrome, representing extremes of the clinical spectrum, are presented. Special emphasis has been directed to the results of the simultaneous studies of alveolo-respiratory and hemodynamic function which were carried out.

The first patient, his diagnosis having been established by lung biopsy, was studied initially shortly after clinical onset of the disease. He was found to have a normal pulmonary artery pressure at rest, but pulmonary hypertension during exercise. Coincident with the development of pulmonary hypertension, a diffusion insufficiency for oxygen appeared. This latter state was defined by development of an abnormally wide alveolar-arterial oxygen tension gradient, in the absence of any fixed intrapulmonary right to left shunt. The same patient was restudied after six months of steroid therapy, at which time he had attained improvement both in subjective clinical symptoms and the radiologic extent of the pulmonary infiltrate. Despite such apparent amelioration of the process, the repeat cardiopulmonary evaluation revealed no alteration of the initial physiologic deficits.

The second patient, whose diagnosis was established at autopsy, was studied in the terminal phase of her illness, one month prior to death. In this instance, pulmonary hypertension and a severe diffusion insufficiency for oxygen were present in the resting state. Both defects were improved when pulmonary blood flow was decreased by the carefully-monitored infusion of a ganglionic blocking agent, hexamethonium.

In both cases, the pulmonary vascular resistance was found to be fixed and elevated.

These findings have led to the conclusion that pulmonary hypertension and diffusion insufficiency for oxygen are a consequence of the same primary disorder; namely, extensive destruction of the pulmonary capillaries, which is reflected by the elevated and fixed pulmonary vascular resistance. In the face of this abnormality, the degree of pulmonary hypertension and diffusion insufficiency for oxygen are directly related to the pulmonary blood flow which the diminished vascular bed is called upon to accept. Both of these functional defects are heightened by increased blood flow and ameliorated when flow is lowered.

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RESUMEN

Se presentan dos casos de síndrome de Hamman-Rich que representan los extremos del especto clínico. Se ha puesto especial empeño en los resultados de los estudios simultáneos alveolo-respiratorios, y, función hemodinámica.

El primer enfermo, en el que se comprobó el diagnóstico por biopsia pulmonar, se estudió poco después del principio clínico de la enfermedad. Se encontró que tenía presión de la arteria pulmonar normal en reposo, pero que había hipertensión pulmonar en el ejercicio. Al mismo tiempo que se desarrolló hipertensión pulmonar apareció insuficiencia de la difusión del oxígeno. Esto último se definió por el desarrollo de un gradiente de tensión arterial del oxígeno anormalmente amplio, en ausencia de cualquiera intercomunicación intrapulmonar fija de derecha a izquierda. El mismo enfermo fué vuelto a estudiar seis meses después de tratamiento con esteroides, cuando obtuvo mejoría tanto en los síntomas clínicos subjetivos como en la extensión radiológica del infiltrado. A pesar de tal mejoría aparente del padecimiento, la evaluación cardiopulmonar no reveló alteración en el déficit fisiológico inicial.

El segundo enfermo, cuyo diagnóstico se estableció por la autopsia, se estudió en la fase terminal de la enfermedad, un mes antes de la muerte. En este caso, la hipertensión pulmonar y la insuficiencia severa de la difusión del oxígeno se observaron en el estado de reposo. Ambos defectos mejoraron cuando el flujo sanguíneo disminuyó por la infusión muy cuidadosa de un agente ganglioplejico: el hexametonio.

En ambos casos la resistencia vascular pulmonar se encontró fija y elevada. Estos hallazgos han conducido a pensar que la hipertensión pulmonar y la insuficiencia de difusión de oxígeno son consecuencia del primer trastorno mismo o sea la destrucción extensa de los capilares pulmonares que refleja por la resistencia vascular pulmonar elevada y fija.

Frente a esta abnormalidad, el grado de hipertensión pulmonar y la insuficiencia de difusión de oxígeno están en relación directa con el flujo sanguíneo que está obligado a recibir el lecho vascular disminuido. Ambos defectos funcionales se acentúan al crecer el flujo sanguíneo y mejoran cuando éste disminuye.

RESUME

Les auteurs présentent deux cas de syndrome de Hamman-Rich, représentant des manifestations extrêmes de la gamme des faits cliniques. Ils mettent en évidence les résultats des études de la fonction alvéolo-respiratoire et de la fonction hémo-dynamique pratiquées simultanément.

Le premier malade, une fois son diagnostic établi par biopsie pulmonaire, fut étudié pour la première fois peu de temps après l'apparition de la maladie. On trouva qu la pression de l'artère pulmonaire était normale au repos, mais une hypertension pulmonaire apparaissait après exercice. Coïncidant avec le développement de l'hypertension pulmonaire, apparut une insuffisance de la diffusion de l'oxygène. Cet état fut limité par le développement d'un degré d'oxygénation alvéolo-artériel anormalement étendu, en l'absence de shunt intrapulmonaire stable de droite à gauche. Le même malade fut étudié de nouveau après six mois de corticothérapie, au bout de laquelle survint une amélioration portant à la fois sur les symp-

tômes cliniques subjectifs et sur les manifestations radiologiques d'infiltration pulmonaire. Malgré cette amélioration apparente, la répétition du bilan cardiopulmonaire ne révéla aucune modification des déficits physiologiques initiaux.

Le deuxième malade, dont le diagnostic fut établi à l'autopsie, fut étudié dans la phase terminale de sa maladie, un mois avant sa mort. Dans ce cas, il existait une hypertension pulmonaire et une insuffisance grave de la diffusion de l'oxygène, au repos. Les deux troubles étaient améliorés lorsque le débit sanguin pulmonaire diminuait par perfusion soigneusement contrôlée d'un agent ganglionnaire de blocage, l'héxamethonium.

Dans les deux cas, on trouva une résistance vasculaire pulmonaire fixée et élevée.

Ces constatations ont amené à conclure que l'hypertension pulmonaire et l'insuffisance de diffusion de l'oxygène sont la conséquence du même trouble primaire; c'est-à-dire la destruction extensive des capillaires pulmonaires, qui est reflétée par la résistance pulmonaire vasculaire fixée et élevée. Devant cette anomalie, le degré de l'hypertension pulmonaire et l'insuffisance de diffusion de l'oxygène sont directement en liaison avec le débit sanguin pulmonaire, qu'est obligé de subir le lit vasculaire diminué. Ces deux troubles fonctionnels sont augmentés par l'accroissement du débit sanguin; ils sont améliorés lorsque le débit s'abaisse.

ZUSAMMENFASSUNG

Es werden 2 Fälle von Hamman-Rich-Syndrom vorgestellt, die Extreme des klinischen Spektrums darstellen. Besonderer Wert wurde auf die Ergebnisse der gleichzeitig vorgenommenen Untersuchungen der Alveolären Respiratorischen und haemodynamischen Funktion gelegt.

Bei dem ersten Kranken war die Diagnose durch Lungenbiopsie gesichert worden, und er wurde zuerst kurz nach seinem klinischen Krankheitsbeginn untersucht. Es ergab sich bei ihm ein normaler Pulmonal-Arterien-Druck in Ruhe, jedoch pulmonaler Hochdruck bei Belastung. Gleichzeitig mit der Entwicklung der pulmonalen Hypertension trat eine Insuffizienz der Sauerstoff-Diffusion auf. Dies werde durch das Auftreten eines abnorm weitzen alveolären arteriellen Sauerstoff-Druckgefälles bei Fehlen irgend eines fixierten Rechts-Links-Shuntsbestimmt. Der gleiche Kranke wurde nach 6 Monate langer Steroid-Therapie wieder untersucht, und er hatte zu diesem Zeitpunkt eine Besserung sowohl hinsichtlich der subjektiven klinischen Symptome erzielt, als auch der röntgenologischen Ausdehnung der pulmonalen Infiltrate. Trotz solcher augenscheinlicher Besserung des Prozesses ergab die wiederholte cardiopulmonale Auswertung keine Änderung der anfänglichen festgestellten physiologischen Defekte.

Der zweite Fall, dessen Diagnose durch Sektion gewonnen wurde, wurde im Endstadium der Erkrankung, ein Monat vor seinem Tod untersucht. In jenem Augenblick bestand ein pulmonaler Hochdruck und eine beträchtliche Insuffizienz der Sauerstoff-Diffusion in Ruhe. Beide Schäden werden gebessert wenn die pulmonale Blutdurchströmung durch sorgfältig begrenzte Infusion einer Ganglien blockierenden Substanz, dem Hexamethonium herabgesetzt wurde.

In beiden fällen ergab sich, dass der pulmonale Gefässwiderstand fixiert und erhöht war. Diese Befunde haben zu der Schlussfolgerung geführt, dass pulmonaler Hockdruck und Sauerstoff-Diffusions-Insuffizienz die Folge ein und derselben Störung sind, nämlich ausgedehnter Destruktion der pulmonalen Kapillaren, die im erhöhten und fixierten pulmonalen Gefässwiderstand zum Ausdruck kommt. Angesichts dieser Abnormalität stehen das Ausmass der pulmonalen Hypertension und die Sauerstoff-Diffusions-Insuffizienz in direkter Beziehung zur Lungendurchblutung, die das verkleinerte Gefässbett übernehmen muss. Diese beiden funktionellen Mängel steigern sich durch Zunahme der Durchströmung und bessern sich, wenn die Durchströmung geringer wird.

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Penetrating Wounds of the Chest

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A wealth of information regarding proper management of penetrating chest injuries in battle casualties has appeared in the surgical literature since World War II and the Korean War. Sanger,[1] Burford, Parker and Sampson,[2] Welch and Tuhy,[3] and others[4] have described experiences during World War II, placing emphasis on early restoration of cardiopulmonary physiology. Rockey,[5, 6, 7] Valle,[8] and Artz, Bronwell and Sako[9] reviewing Korean War experiences, gave credit to more rapid medical evacuation, early definitive surgery, blood volume replacement and antibiotics, in achieving marked reduction in mortality and morbidity statistics. Despite the frequency of these wounds in civilian practice, little attention has been given this subject in post-war literature. Farringer and Carr[10] reviewed 251 cases of penetrating wounds of the chest seen between 1946 and 1950. They pointed out that with the proper use of our present methods of treatment few patients with penetrating wounds of the chest, seen in civilian practice, expire as a result of their wounds. They stressed the importance of preventing the sequela of these injuries such as empyema or a fibrous encasement of the lung.

The purpose of this paper is to review 101 cases of penetrating and perforating chest wounds treated in private practice and at the Veteran's Administration Hospital, Jackson, Mississippi from May, 1951 through August, 1957, assessing their treatment and applying accepted thoracic surgical principles.

Analysis of Cases

Of the 101 cases, 81 were Negro and 20 were white. The ages ranged from six to 66 years. The weapons or instruments producing the injuries are listed in Table I.

Initial Management

On admission, patients were placed in the semi-Fowler position to permit easier respiration; clothing was removed; and the wounds carefully surveyed. Sucking wounds were closed by vaseline gauze dressing. If the underlying lung had a significant air leak, a catheter was placed in the chest through the wound and connected to a water trap until the patient's general condition could be stabilized for definitive surgery.

Blood pressure, pulse, respiration and hematocrit were checked frequently. When possible, anti-shock therapy was instituted even before the classical picture of shock developed. The causes of shock, blood loss, pain and anoxia, were eliminated as soon as possible. Transfusions were given as indicated to restore normal blood volume. Intercostal nerve blocks were given to relieve painful rib fractures. All patients were given adequate but not depressing doses of narcotics. Cardiorespiratory imbalance

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due to partial lung collapse, mediastinal shift or obstruction of the airway was corrected to eliminate anoxia due to these causes. Oxygen was given by nasal catheter at the rate of eight liters per minute.

Prophylactic antibiotics and anti-tetanus immunization were given.

Gastric suction was employed for low chest wounds with the possibility of associated abdominal injuries.

Posterior anterior and lateral chest roentgenograms were obtained as soon as practicable, either in the X-ray Department or by a portable unit in the Emergency Room. These were a primary aid in further assessment of the patient's injury and served as indicators for more definitive therapy. If air and fluid were not noted in the pleural space, the film was repeated in 24 hours, for on occasions these were noted to develop after admission.

Once the general condition of the patient had stabilized, wounds could be debrided and closed.

Aspiration and Catheter Drainage

Thirty-six patients with a minimum hemothorax and/or pneumothorax were not aspirated or drained. Thoracentesis was done on all cases in which the fluid and/or air would not absorb in a few days. This was not considered an emergency procedure except in those cases with tension pneumothorax or extensive hemothorax interfering with cardiorespiratory function. The first thoracentesis was usually done in the first 24 hours and repeated daily until the lung was completely expanded. As much fluid and air as possible up to 1000 cc. was removed at a time without producing symptoms. Coughing, dyspnea, tachycardia, or tightness in the chest were considered indications to terminate the procedure. Thoracentesis was done in 51 cases.

In 17, persistent air leak necessitated insertion of a catheter into the pleural cavity. Under local anesthesia a no. 24 F catheter was inserted in the second intercostal space anteriorly and connected to a water trap permitting gradual decompression as the lung expanded. Catheter drainage was used early in all cases of tension pneumothorax.

Open Thoracotomy

The following were considered as indications for open thoracotomy:

1. Intrapleural bleeding is an indication for thoracotomy if severe and persistent. Bleeding of this severity in patients who live to reach the hospital usually comes from an intercostal or internal mammory vessel.

TABLE 1

WEAPONS OR INSTRUMENTS PRODUCING INJURIES

Type	Number
Knife	50
Rifle and Pistol	27
Shotgun	12
Ice Pick	11
Automobile Radio Antenna	1
TOTAL	101

2. Massive clotted hemothorax in a recently injured patient usually indicates damage to a large vein or atrium. Hemorrhage is likely to be profuse and blood loss will exceed blood replacement until the bleeding point is controlled surgically. When this condition exists, emergency surgery is indicated before the patient's condition has been stabilized.

3. Cardiac tamponade is diagnosed by distention of neck veins while in the sitting position, rapid thready pulse, decrease in pulse pressure, distant cardiac sounds and loss of cardiac pulsation as noted on fluoroscopy. Pericardicentesis is performed and if the condition recurs the bleeding point is controlled at surgery.

4. Sucking wounds require modified thoracotomy usually through the wound, debridement, pleural lavage and closure with drainage.

5. Large wounds of the trachea or bronchi require open thoracotomy. Bronchial wounds usually cause severe air leak and diagnosis is no problem. Wounds of the trachea may produce mediastinal emphysema or unexplained subcutaneous emphysema in the neck. Voice changes are sometimes seen. Respiratory difficulty is usually apparent.

6. Suspected esophageal lacerations or perforations should be explored as soon as the patient's condition is stabilized. The diagnosis of esophageal damage may be suspected when the course of the projectile is such that it may have involved this organ. The presence of mediastinal emphysema and the demonstration of a defect by barium swallow will establish the diagnosis. We believe that exploration should be carried out when involvement of the esophagus is strongly suspected. If not repaired, esophageal leak will lead to severe mediastinitis.

7. Intrapleural foreign bodies, 1.5 cm. or longer, should be removed. The wound of entrance should be debrided at the same time and closed with drainage. When accessible, we believe that large intrapulmonary foreign bodies should also be removed. We have recently had two cases

FIGURE 1

FIGURE 2

Figure 1: Roentgenogram of chest taken in 1957 showing reaction about foreign body which had been present since 1918.—*Figure 2* (Case 1): Roentgenogram on admission reveals multiple shot in left chest. There is fracture of fourth and fifth ribs on the left. There is pneumothorax on the left and shift of mediastinum to the right.

of pulmonary abscess forming at site of retained foreign body which had been present since World War II. Both were successfully removed. A third patient who had a large retained shrapnel dating from World War I had advanced unilateral bronchiectasis and pulmonary fibrosis secondary to infection. Soon after his injury in 1918, he developed empyema which was cured by open drainage. He has had hemoptysis on occasions since then. Roentgenogram of the chest made in 1957 revealed marked reaction to the foreign body (Figure 1). Bronchograms revealed localized bronchiectasis.

8. Injury or suspected injury to the diaphragm and abdominal viscera is an indication for exploration. In those cases where thoracotomy is indicated according to the above criteria, either thoracotomy or thoracoabdominal incision is done. For a low thoracic wound perforating the diaphragm with minimal or no damage to the lung, laparotomy is performed and the diaphragm repaired from below. The pleural cavity is treated by thoracentesis or catheter drainage as indicated.

Of the 101 cases treated, five required early thoracotomy using these indications. Eight others with lower thoracic and abdominal injuries were treated by laparotomy.

Decortication

Three patients with empyema required surgical decortication. Two were done three weeks after injury with good results. The third came in with empyema ten months after injury. The empyema was cured by decortication, open drainage and thoracotomy, but pulmonary function was definitely impaired.

TABLE II

COMPLICATIONS

Type	Number
Hemothorax	69
Pneumothorax	62
Subcutaneous Emphysema	16
Shock	15
Atelectasis	11
Traumatic Pneumonitis	10
Rib Fractures	6
Injury of Diaphragm and Abdominal Viscera	6
Empyema	4
Hemopericardium or Pericarditis	3
Pleural Effusion	3
Pulmonary Edema	1
Lung Abscess	1
Perforation of Atrium	1
Diaphragmatic Hernia	1
Spinal Cord Injury	1
Bronchopleurocutaneous Fistula	1

Seven with clotted hemothorax were successfully treated by chemical decortication using streptokinase and streptodornase.

Complications

The complications are listed in Table II. The most common were hemothorax, pneumothorax, subcutaneous emphysema, shock, atelectasis and traumatic pneumonitis. The single case of bronchopleurocutaneous fistula was in a patient admitted 12 days after receiving a butcher knife stab wound of the right chest.

Deaths

There were eight deaths in this series. Two died within 25 minutes of admission before definitive therapy could be instituted. One died four hours after admission as a result of perforating wound of the right atrium and a large tear of pericardium associated with massive clotted hemothorax. He did not respond to therapy for shock and was never considered in condition for surgery. It is possible that emergency thoracotomy could have saved his life even though his condition was poor from the time of admission to death. Three died several days after admission with lower nephron nephosis secondary to shock. All three had extensive injuries. One admitted in an extremely toxic condition, two and a half days after severe injuries of the left upper chest, developed pulmonary edema soon after admission and died within less than 24 hours. One who had cirrhosis of the liver developed hepatic coma and died 10 days after admission. His chest injuries were inflicted while he was in another hospital and did not seem to affect the course of his disease.

Illustrative Cases

Case 1: B. I. T., a 24 year old man, was admitted to the hospital several hours after being shot in the left chest at close range with a shotgun. On admission, he

FIGURE 3

FIGURE 4

Figure 3 (Case 2): Roentgenogram of chest taken on admission. Metal clips in left supraclavicular region mark the wound of entrance. There is hemopneumothorax on the left.—*Figure 4* (Case 3): Roentgenogram of the chest does not reveal evidence of pneumothorax. There is free air under the diaphragm.

did not appear to be in shock. His blood pressure was 140/80. He appeared to breathe shallow and rapid. His mediastinum was shifted to the right. On the left extending between the mid-axillary line and the anterior axillary line there was a 10 x 15 cm. defect in the skin with some extension of the wound into the pleural cavity in two small areas. There was marked damage to the pectoral and intercostal muscles in this area. Many small perforations associated with powder burns of the skin were noted throughout the left upper chest. Roentgenogram (Figure 2) revealed extensive fractures of the fourth and fifth ribs on the left. The left lung was atelectatic. A thoracotomy tube was placed through the wound into the chest and connected to a water trap. After he had adequately stabilized, thoracotomy was done. In many sucking wounds of the chest on adequate debridement can be carried out through the wound. In this case, however, we feit that the tissues of the chest wall and the left lung were so badly damaged that a classical thoracotomy incision should be done entering the chest through the bed of the fourth rib. Fragments of the fifth rib were removed. The chest wall was debrided requiring removal of a large amount of muscle tissue and skin. This created a large defect in the anterolateral aspect of the chest wall. A large part of the left lung was badly contused. A 3 x 5 cm. area of lung tissue was destroyed. The lung tissue was debrided and closed with 00 chromic catgut. The pleural cavity was irrigated and drained. It would have been difficult

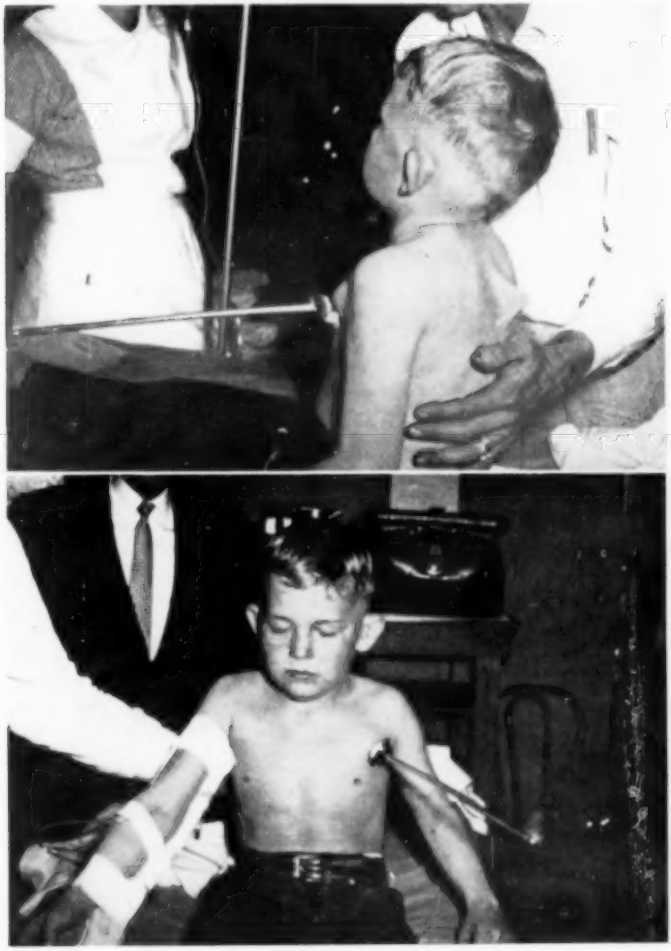

FIG. 5

FIG. 6

Figures 5 and 6 **(Case 4):** Photographs of patient with discarded automobile radio antenna perforating left chest.

to close the chest wall defect without the classical thoracotomy periscapular incision. In closure, the scapula was permitted to fall forward. This made it possible to close the defect in the anterolateral aspect of the chest wall. The postoperative course was uneventful except for febrile episode for the first two postoperative days. He had no postoperative air leak. He was discharged on the 10th postoperative day.

Case 2: H. T., a colored man, was stabbed in the left supraclavicular region the night before admission. The wound was closed with skin clips by his private physician. Though he had no chest pain or dyspnea, a roentgenogram (Figure 3) the following day revealed hemopneumothorax which responded to thoracentesis. This case demonstrates chest injury as a complication of supraclavicular wounds.

Case 3: L. T., a colored man age 23 years, was admitted about 36 hours after being stabbed in the left chest. He was admitted to another hospital, treated until it became evident he would need surgery, and then transferred to the Veteran's Administration Hospital.

On admission, temperature was 101°F.; pulse 110 per minute, and blood pressure 124/90 mm. mercury. There was a ½ inch long laceration in the left seventh intercostal space in the mid-axillary line. There was generalized abdominal tenderness and board like rigidity of the abdomen. Roentgenogram of the chest (Figure 4) revealed good expansion of both lungs with free air under the diaphragm.

Exploratory laparotomy was performed on the day of admission and he was found to have a laceration of the diaphragm and anterior wall of the stomach. They were repaired and he recovered.

Case 4: T. B., a six year old white boy, was admitted to the Mississippi Baptist Hospital about an hour after he fell while playing with a discarded automobile radio antenna perforating the left chest (Figure 5 and 6). He had tried but failed to remove it. On admission, he was anxious and tense but his general condition was fair. His blood pressure was 110/70, and pulse 130. A 2½ foot metal rod entered the left chest through the third intercostal space in the anterior axillary line. It passed obliquely to the right and posteriorly and emerged through the skin just to the right of the midline at about the fifth intercostal space level. There was decrease in expansion of the left chest. Both lungs were normally resonant to percussion and rales were not heard.

Hemogram: red blood count was 5,000,000; hemoglobin 13.7 gms.; white blood count 19,200.

Roentgenogram of the chest, posteroranterior and lateral views, revealed a metal rod extending into and through the left hemithorax. The point of entrance was between the third and fourth ribs in the anterior axillary line and the point of exit was between the fifth and sixth ribs on the left posteriorly just adjacent to the spine. There was no evidence of fluid or air in the left pleural space. There was no pulmonary reaction about the rod (Figure 7).

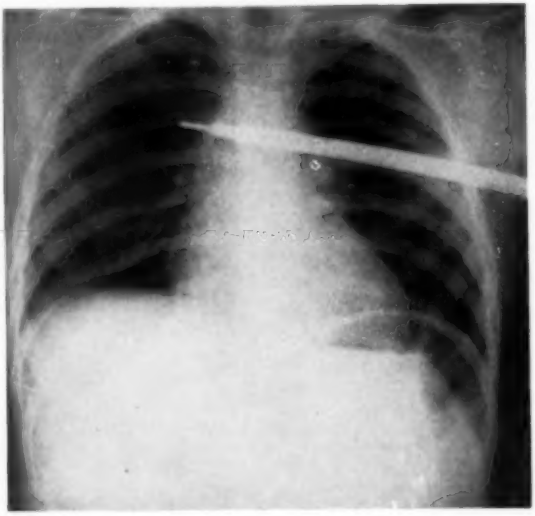

FIGURE 7 (Case 4): Roentgenogram of chest with discarded automobile radio antenna perforating left chest.

Thoracotomy was performed and the rod was removed. It traversed the upper lobe at the junction of the upper lobe with the lingula. It passed through the superior segment of the lower lobe. There was little bleeding but there was moderate air leak from the lung when the rod was removed. The wounds were repaired. The chest was closed with drainage and he had an uneventful recovery. He was discharged on the eighth postoperative day.

SUMMARY

1. One hundred and one penetrating and perforating chest wounds seen in civilian practice since 1952 have been reviewed and factors common to the management of the acute phases and complications of these problems have been discussed.

2. Aspiration of hemothorax is preferable to catheter drainage.

3. In pneumothorax, the lung may be expanded by thoracentesis; however, in case of persistent air leak shown by increase in pneumothorax, progression of subcutaneous emphysema or by tension pneumothorax catheter drainage is used.

4. Of the 101 cases treated 36 did not require surgical procedure except the local care of the wound, antibiotics and tetanus immunization. Fifty-one required drainage by thoracentesis. Catheter drainage was used in 17 cases. Eight required thoracotomy including three decortications. Laparotomy was performed in eight cases.

RESUMEN

1. Ciento una heridas penetrantes de tórax vistas en la práctica civil desde 1952, se han revisado y se discuten los factores comunes en el tratamiento en las fases agudas y en sus complicationes.

2. Es preferible la aspiración en lugar de la canalización con cateter.

3. En el neumotórax, el pulmón puede expanderse por toracentesis; sin embargo, en caso de persistente escape de aire como lo muestra el aumento del neumotórax, el crecimiento del enfisema subcutáneo a presión, se usa la canalización por el cateter.

4. De los 101 casos tratodos, 36 no requirieron operación quirúrgica con excepción del cuidado local de la herida, los antibióticos y la inmunización para el tétanos. Cincuenta y uno requirieron canalización por toracentesis. Se usó la canalización por cateter en 17. Ocho requirieron toracotomía incluyendo tres de decorticación. Se llevó a cabo laparotomía en ocho casos.

RESUME

1. Les auteurs font l'exposé de 101 observations de blessures thoraciques pénétrantes et perforantes vues en clientèle privée depuis 1952. Ils discutent les facteurs communs du traitement des phases aiguës et des complications de ces conditions.

2. L'aspiration de l'hémothorax est préférable au drainage par sonde.

3. Dans le pneumothorax, le poumon peut être mis en expansion par aspiration; cependant, en cas d'anoxie persistante, prouvée par l'augmentation du pneumothorax, la progression de l'emphysème sous-cutané ou par la tension du pneumothorax, on peut utiliser le drainage par sonde.

4. Sur 101 cas traités, 36 ne nécessitèrent pas d'intervention chirurgicale, et on put se con tenter des soins locaux de la blessure, des antibiotiques, et de la vaccination antitétanique. 51 cas nécessitèrent un drainage par ponctions. Le drainage par sonde fut utilisé dans 17 cas. Huit cas nécessitèrent une thoracotomie et parmi eux il y eut trois décortications. On pratiqua une laparotomie dans huit cas.

ZUSAMMENFASSUNG

1. Übersicht von 101 penetrierenden und perforierenden Thoraxwunden aus einer zivilen Praxis seit 1952 mit Besprechung der Kompilkationen diser Schwierigkeiten und der Faktoren, die gewöhnlich während der Behandlung der akuten Phase vorkommen.

2. Die Punktion und Absaugung eines Haemothorax ist der Drainage mittels Katheter vorzuziehen.

3. Bei einem Pneumothorax kann die Lunge durch eine Thorakozentese zur Wiederausdehnung kommen; es wird jedoch in Fällen persistierender Luftdurchlässigkeit, wie sie sich zeigt an einer Vergrösserung des Pneumothorax, Zunahme des subkutanen Emphysems oder an einem Spannungs-Pneumothorax, eine Katheter-Drainage benutzt.

4. Von den 101 behandelten Fällen benötigten 36 keine chirurgischen Massnahmen abgesehen von der lokalen Wundversorgung, Antibiotizis und Tetanus-Injektion. 51 benötigten Drainage durch Thorakozentese. Drainage mittels Katheter wurde bei 17 Fällen gebraucht 8 erforderten eine Thorakotomie einschliesslich Dekortikationen. Eine Laparatomie wurde in 8 Fällen vorgenommen.

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SECTION ON CARDIOVASCULAR DISEASES

Diagnosis of Ventricular Septal Defects*

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During the past 10 years the story of ventricular septal defect has been rewritten. Formerly it was generally regarded as a benign lesion, recognized on the basis of certain physical signs in the absence of cardiac enlargement or symptomatology.[1, 2] More recently it has come to be appreciated as a serious cardiac anomaly associated with moderate or marked cardiomegaly and responsible for considerable cardiac disability and premature death.[3, 4] It is fortunate that just as the true character of ventricular septal defect became apparent, that a happy ending to the story was made possible by the development of surgical techniques to close the defect.[5, 6]

Diagnosis of this condition has also changed during the past decade. The characteristic coarse thrill and long, loud harsh systolic murmur over the body of the heart, so well described by Roger,[7] remain important aids to the diagnosis, but there are additional criteria which, when met, make the diagnosis much more secure.

When attention was paid primarily to the precordial systolic murmur in a relatively asymptomatic individual, it was found that the diagnosis of ventricular septal defect was made much too often. Some of these patients were shown subsequently to have instead, mild pulmonic or aortic stenosis, auricular septal defect, idiopathic dilatation of the main pulmonary artery, mitral insufficiency, or an innocent "functional" murmur.

In contrast, when patients were examined who had great cardiac enlargement and marked limitation of activity or even congestive heart failure, the diagnosis of ventricular septal defect was rarely entertained.

Although there are undoubtedly many patients who conform to Roger's description of striking physical signs in the absence of symptoms, I wish to emphasize diagnosis and differential diagnosis of the large number of patients with disability who will be candidates now or in the future for corrective surgery. While some features are characteristic of this anomaly, regardless of age of the patient, others are peculiar to infancy, to childhood or to adult life. The diagnostic aspects of this malformation at these three age periods will be considered in turn.

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Infancy

It is during infancy that morbidity and mortality produced by ventricular septal defects are the highest.[8] The critical condition of the babies makes more hazardous or difficult the performance of certain tests, such as cardiac catheterization, that could be carried out unhesitatingly in older patients. It is therefore desirable to derive as much information as possible from each step of the examination.

The history of babies with this malformation often includes some or all of the following features:

Birth weight low for gestational age
Failure to gain weight
Feeding difficulty: poor suck
easy fatigue
Respiratory distress: labored respiration
cough
Susceptibility to respiratory infections
Excessive perspiration
Aphonic cry

These symptoms are not limited to babies with ventricular septal defect, however, but are characteristic of infants with any large left-to-right

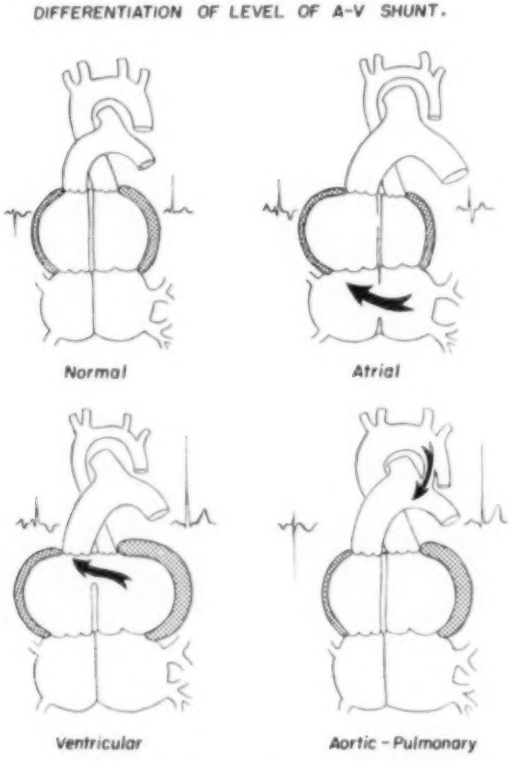

FIGURE 1: Schematic representation of chamber size, wall thickness, and relative size of the great vessels when a left-to-right shunt occurs at the atrial, ventricular, and aortic-pulmonary levels.

shunt. Such a history suggests the possibility of ventricular septal defect; other information is necessary to define the site of the shunt. The information we seek is illustrated schematically in Figure 1. Here are depicted the changes in chamber size and wall thickness and in the relative or actual size of the great vessels that result when a left-to-right shunt occurs at the atrial, ventricular, or aortico-pulmonary level. In all, the main pulmonary artery and its branches are enlarged, but the size of the left ventricle and of the aorta help to differentiate the level of the shunt.

Thus in an atrial septal defect or in anomalous pulmonary venous return to the right atrium or its tributaries, where the shunt occurs proximal to the atrioventricular valves, the right atrium and ventricle accommodate a great volume of blood and are dilated. The left ventricle and aorta receive much smaller amounts of blood and are not enlarged; indeed, they may be smaller than normal. The electrocardiogram shows a conduction disturbance over the right ventricle but no hypertrophy pattern.

In contrast, when the shunt of blood occurs distal to the atrio-ventricular valves, there is enlargement and hypertrophy of the left ventricle. If the shunt is at the ventricular level, then the aorta receives only a fraction of the blood that entered the left ventricle and in relation to the pulmonary artery, the aorta is small. The electrocardiogram shows a pattern of left ventricular hypertrophy, often with a partial right bundle branch block over the distended right ventricle.

When the left-to-right shunt takes place at the aortic-pulmonary level, either through a patent ductus or a defect in the aortic septum, the increased blood volume returning from the lungs passes through both left heart chambers and the aorta before it divides into the pulmonary and systemic circulations. The aorta, therefore, is enlarged in addition to left ventricular and often auricular enlargement when the shunt occurs at the aortic-pulmonary level. The electrocardiogram shows left ventricular hypertrophy.

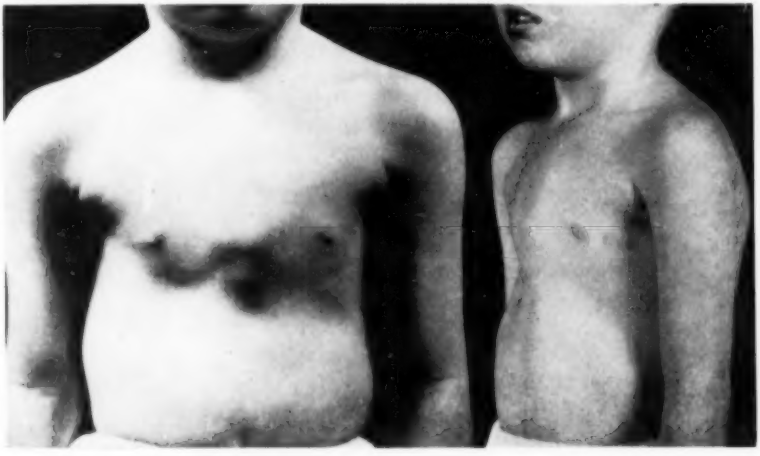

FIGURE 2A FIGURE 2B

Figure 2: Frontal and oblique views of thoracic deformity in child with large ventricular septal defect. Note retraction of suprasternal notch and grooving of ribs as well as the precordial bulge to the left of the sternum and in the anterior axillary line.

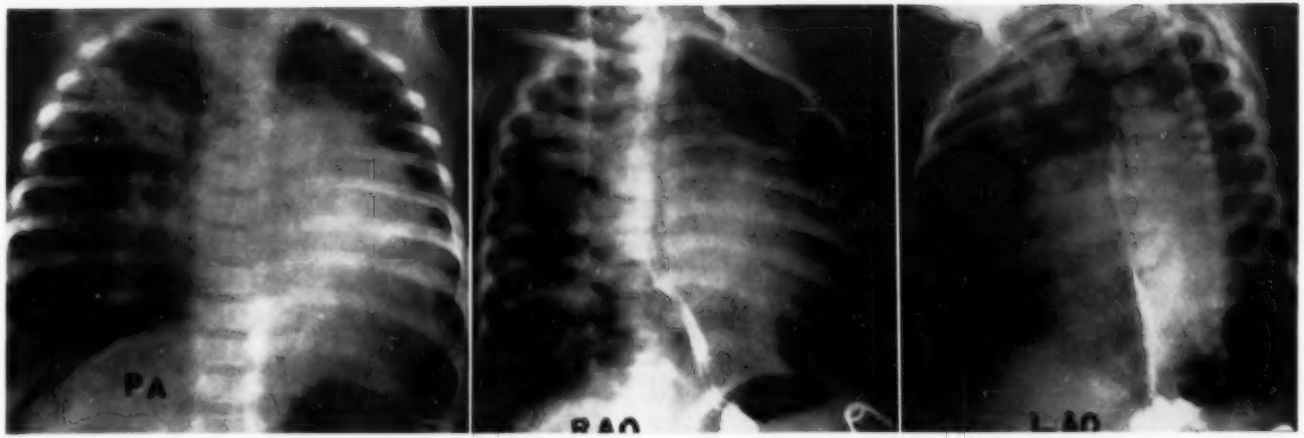

FIGURE 3A FIGURE 3B FIGURE 3C

Figure 3: Roentgenogram of chest in infant who died at age four months because of a large ventricular septal defect. Cardiac enlargement affects all four chambers. The main pulmonary artery is prominent and the branches engorged. The aorta is small.

Thus the size of the left ventricle helps to differentiate shunts proximal and distal to the atrioventricular valves, and the size of the aorta helps to localize the shunt proximal or distal to the aortic valve. These relationships are applicable to shunts of moderate or large size at all ages but are particularly helpful in infancy where the murmur of the shunt at any of the three levels may be only systolic in time.

Some information concerning the status of the pulmonary circulation and the size of the left ventricle is available from physical examination. One sees an undernourished infant without cyanosis but with rapid or labored respirations. There are often retractions at the suprasternal notch and at the level of the diaphragms. The heart is enlarged and over-active, and there is a precordial bulge just to the left of the sternum overlying the right ventricle and the pulmonary artery, and another at the apex overlying the left ventricle, in the anterior axillary line around the fifth and sixth interspaces. If the baby survives, a fixed thoracic deformity results from the combination of labored respirations and enlargement of the right and left sides of the heart. This is seen in Figure 2.

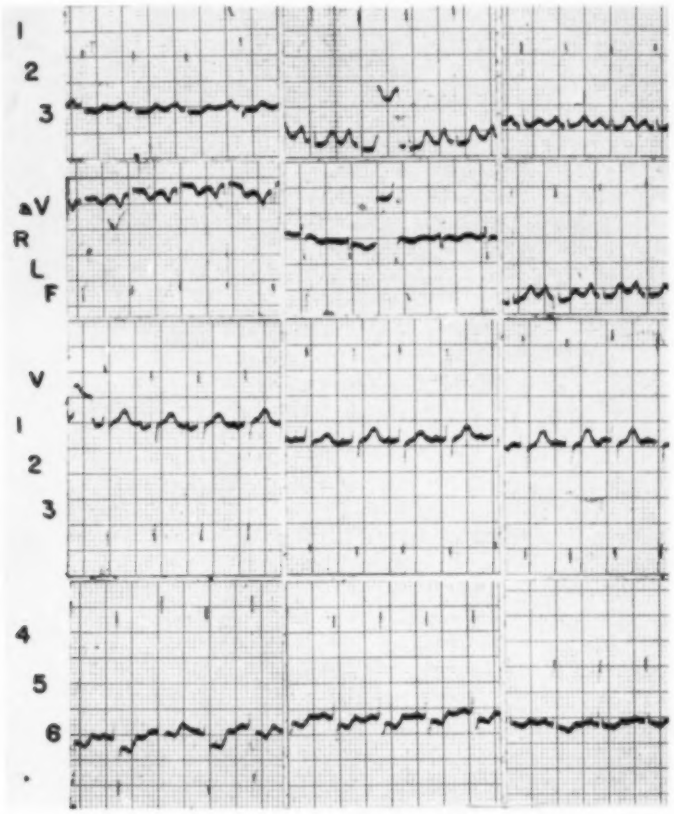

FIGURE 4: Electrocardiogram of infant in heart failure because of ventricular septal defect. There are tall R waves in the left precordial leads, aVF, and leads 2 and 3. Abnormal T waves are present; they are negative in left precordial and positive in right precordial leads.

A coarse systolic thrill is palpable in the left parasternal area in the third to fifth interspaces, and a long, loud, harsh systolic murmur is heard with maximal intensity in the same area. It is audible all over the precordium and in the lung fields. A short mid-diastolic murmur of relative mitral stenosis can often be heard at the apex. The pulmonary second sound may be split and is considerably louder than the aortic second sound. Peripheral pulses may be of small volume, but the arm and leg pressures are normal. If congestive failure is present, rales in the lungs and enlargement of the liver will be found.

Laboratory studies strengthen the diagnosis. Fluoroscopy shows engorged lung fields and a prominent main pulmonary artery and branches which exhibit expansile pulsations of wide amplitude. The aorta is small in comparison, and in the left anterior oblique view seems normal in posi-

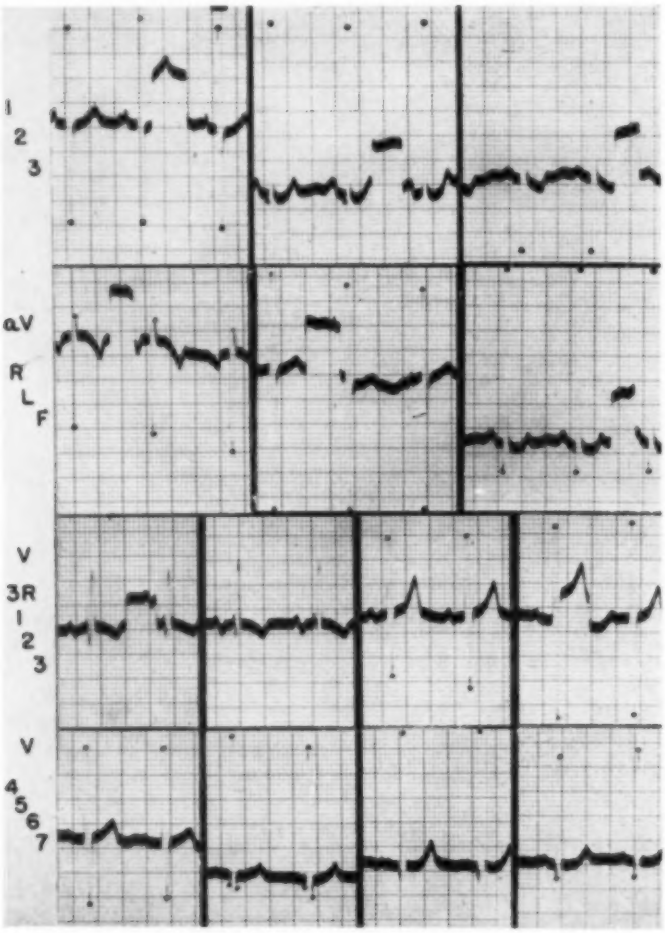

FIGURE 5: Electrocardiogram of young child with ventricular septal defect shows wide, biphasic RS in lead 1 with evidence in V1 of a partial right bundle branch block and in V5 and 6 of left ventricular hypertrophy. T waves in left precordial leads are high. There are both peaking and notching of broad P waves.

tion. Cardiac enlargement is predominantly left-sided though it involves all four chambers. A typical chest roentgenogram is seen in Figure 3.

The electrocardiogram reveals left ventricular hypertrophy. In contrast to older patients, often there is left ventricular "strain" with reversal of the normal T wave pattern in the precordial leads and inverted T waves with depressed ST segments in leads 1 and 2 (see Figure 4). In addition, some patients show an incomplete right bundle branch block in the right precordial leads, while others show combined right and left ventricular hypertrophy. Under these circumstances, there is often a biphasic RS of wide amplitude in lead 1 (Figure 5). Broad, notched P waves reflect the left auricular enlargement, and high, peaked P waves indicate enlargement of the right auricle (Figure 5).

Diagnosis is usually possible on the basis of the composite findings just listed. Corroboratory evidence is best obtained by cardiac catheterization, where a step-up in oxygen content is observed in the samples from the right ventricle with no further increase in oxygenation in the pulmonary artery. In a baby such as we have been considering, the right ventricular and pulmonary artery systolic pressures approach or equal the systemic systolic pressure. "Pulmonary capillary" pressure is usually slightly elevated. The pulmonary blood flow is two or three times as large as the systemic blood flow. There is usually no right-to-left shunting demonstrable by peripheral oxygen unsaturation or dye-dilution curves. (See Case 1 in table).

Angiocardiography in infants with ventricular septal defects is often of little aid in diagnosis.[9] With multiple exposures at high speed a filling defect may be noted in the right ventricle as a jet of unopacified blood from the left heart traverses the defect. Occasionally the shunt may be temporarily reversed and some of the contrast agent can be seen in the aorta at the time of pulmonary arterial filling. Figure 6 illustrates this event in a four month old baby in heart failure who weighed 2360 gm. at the time of the study. At her death at the age of 5 months there was a 2.5 cm. defect in the membranous portion of the ventricular septum. The position of the great vessels was normal, and the ductus was obliterated.

Selective angiocardiography may demonstrate the filling defect or shunt

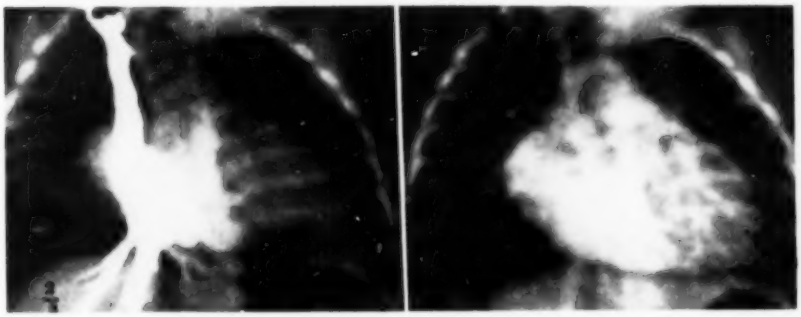

FIGURE 6: Angiocardiogram of 2360 gm. infant at age four months. Contrast material at ⅔ second fills the large right atrium and ventricle and at 1⅓ seconds can be seen not only in the large pulmonary artery but in the smaller aorta as well. At postmortem examination a ventricular septal defect 2.5 cm. in diameter was the sole anomaly.

reversal more precisely. Retrograde aortography to exclude or outline an aortico-pulmonary shunt may be helpful if the diagnosis is still in doubt.

Childhood

Diagnosis of this congenital malformation during the childhood years is easier than in infancy. The same diagnostic features are present, but the patient is less fragile and the symptoms are less extreme. Growth retardation is still evident, but the patient may complain of very little limitation of activity by easy fatigue or shortness of breath despite a surprising degree of cardiac enlargement. Heart failure is much less common now, and if digitalis had been required in infancy, it is usually no longer needed in childhood. Respirations are easy at rest, but grooving of the thoracic cage at the level of the diaphragms remains, as does the precordial bulge.

Physical findings and laboratory data are like those in infancy except that in the electrocardiogram the T waves are positive and tall in the left ventricular deflections (refer to Figure 5). At cardiac catheterization the pulmonary hypertension is due primarily to the large left-to-right shunt. Pulmonary vascular resistance is only moderately increased (around 200-300 dynes/min./cm.$^{-5}$) and "pulmonary capillary" pressure at the upper limits of normal or slightly increased (Case 2 of table).

Adolescence and Adulthood

In the adolescent and young adult the signs of a ventricular septal defect may be subtler still, for at least two reasons. First, there are proportionately more patients with small defects and few hemodynamic changes, and second, there is a group of patients where pulmonary vascular disease alters the picture of ventricular septal defect, either by damping or reversing the left-to-right shunt. In either event, the patient is now well nourished and the precordial bulge less obvious. Symptoms if present consist of shortness of breath and of fatigue on exertion.

For the first group with a small septal defect, diagnosis rests chiefly on the characteristic thrill and murmur and on the step-up in oxygen content at the ventricular level on cardiac catheterization (Case 3 in table).

For the second group with pulmonary vascular disease, certain clinical features suggest a ventricular septal defect but diagnosis depends primarily on cardiac catheterization and angiocardiography. In these patients where structural changes in the pulmonary arterial tree increase the resistance to the flow of blood into the lungs, the left-to-right shunt decreases and, in time, disappears, to be replaced by a right-to-left shunt. The capacity and the work of the left side of the heart are reduced, and the left atrium and left ventricle are no longer enlarged. Instead, the work of the right ventricle is increased. Enlargement of the right ventricle is demonstrable radiographically and hypertrophy of its wall is demonstrated electrocardiographically. The aorta is no longer small and may even become enlarged. In the left anterior oblique position the aorta may appear to arise more anteriorly than usual.

Signs of marked pulmonary hypertension are present and include a loud, banging pulmonary second sound which is sometimes followed by a soft blowing diastolic murmur of pulmonary insufficiency. The impulse imparted by the snapping closure of the pulmonary valve may be palpated

RESULTS OF REPRESENTATIVE RIGHT HEART CATHETERIZATION STUDIES DURING INFANCY, CHILDHOOD, AND ADOLESCENCE OR ADULTHOOD

		Oxygen Content (Vol. Per Cent)								Pressures in Mm. of Hg.					
Case	Age	BA or FA	PA	RV Outflow	RV Inflow	RA	SVC	IVC	Per Cent Oxygen Saturation FA or BA	BA or FA S/D (Mean)	PA	RV	"PC"	P. B. F.* S. B. F.	P. V. R.** in Dynes Sec./Cm.$^{-5}$
1. J. W.	8 mos.	14.5	11.6	13.25	12.08	7.84	....	7.35	95	85/40 (66)	80/30 (62)	80/3	....	2.3	144
2. S. H.	8 yrs.	19.5	16.4	16.6	14.5	13.8	13.1	....	96.9	119/59 (85)	46/14 (32)	43/5	13	2.3	304
3. P. C.	16 yrs.	20.6	17.1	18.1	15.1	15.3	14.8	....	97.9	92/70 (80)	19/9 (13)	18/2	4	1.6	139
4. G. F.	14 yrs.	16.9	11.8	12.0	....	10.0	9.9	7.7	82.3	102/70 (79)	102/71 (79)	101/5	....	1.2	3608

*Ratio of Pulmonary Blood Flow to Systemic Blood Flow.
**Pulmonary Vascular Resistance.

through the chest wall. Radiographically the main pulmonary artery and both branches in the hilar regions are enlarged, but they show minimal intrinsic expansile pulsations and the vascular markings in the peripheral lung fields are scanty.

When this stage is reached, the characteristic thrill and murmur described by Roger may no longer be present. Differential diagnosis of this condition from atrial septal defect with pulmonary hypertension or primary or secondary pulmonary hypertension with right-to-left shunting through a patent foramen ovale requires cardiac catheterization and often angiocardiography.

On cardiac catheterization the pressures in the pulmonary artery are in the range of systemic pressure and the calculated pulmonary vascular resistance is greatly elevated. Shunts through the ventricular defect at this time may be quite variable; their detection is best accomplished by analysis of dye curves and oxygen values. There may be bidirectional shunting, as in Case 4, with a step-up in oxygen content at the ventricular level and a reduction of oxyhemoglobin saturation in the brachial and femoral arteries. No shunt in either direction may be detected at rest but exercise may bring out a veno-arterial shunt. Injection of Evans blue dye into the right auricle, ventricle, or pulmonary artery and analysis of circulation times and dye dilution curves is valuable in detecting the site of shunting in the patient who is transiently or persistently cyanotic.

Angiocardiography often demonstrates a right-to-left shunt in the patient with pulmonary hypertension and actual or impending shunt reversal. Early opacification of the aorta at the time of right heart and pulmonary artery filling, before contrast agent appears in the left auricle or ventricle, gives evidence of a defect at the ventricular level.

In light of experience to date, the diagnosis of ventricular septal defect at this stage automatically places the patient in the inoperable group.[12, 13]

Thus far we have considered the diagnosis of ventricular septal defect as the only cardiac anomaly. When a large defect is associated with certain other conditions, such as mild to moderate pulmonary stenosis or coarctation of the aorta, the presence of the ventricular defect can usually be recognized by the findings already described, and the additional lesion recognized by its characteristic signs. When it is associated with severe pulmonary stenosis, the syndrome we identify as the tetralogy of Fallot is usually easy to diagnose. When other lesions occur together with a ventricular septal defect, diagnostic acumen may be greatly taxed to sort out the pieces.

It is good policy, once the diagnosis of ventricular septal defect is established, to re-examine the data to see if any atypical feature suggests the presence of an additional anomaly. This will sometimes save the cardiologist the embarrassment of having the cardiac surgeon find a coexistent and unsuspected auricular septal defect or silent patent ductus arteriosus.

SUMMARY

Diagnosis of an uncomplicated ventricular septal defect is based on the presence of a coarse systolic thrill and long, harsh systolic murmur maximal in the fourth and fifth left parasternal spaces of a non-cyanotic individual who shows evidence of left ventricular hypertrophy and prominence

of the pulmonary artery without enlargement of the aorta. Cardiac catheterization affords valuable documentation of the size of the shunt, the cardiac burden, and the hemodynamic status of the pulmonary vascular bed. Angiocardiographic techniques assist in diagnosis by demonstration of a right ventricular filling defect during right heart opacification or by premature opacification of the aorta if the shunt is reversed.

Acknowledgment: Cardiac catheterizations on patients 2-4 were performed in the Cardiopulmonary Laboratory under the direction of Dr. Daniel S. Lukas. Angiocardiographic studies were performed by Dr. Israel Steinberg and his associates.

RESUMEN

El diagnóstico del defecto septal ventricular no complicado se basa en la presencia de un thrill rudo sistólico y un soplo áspero largo sistólico con foco en el cuarto espacio paraesternal izquierdo en individuos no cianóticos que tienen evidencia de hipertrofia ventricular y promonencia de la arteria pulmonar sin crecimiento de la aorta.

La cateterización cardiaca provee de información valiosa sobre el tamaño de la intercomunicación, la carga cardiaca, y el estado hemodinámico del lecho vascular del pulmón. Las técnicas angiocardiográficas ayudan al diagnóstico por la demostración de un defecto de llenado del ventrículo derecho durante la opacificación del corazón derecho o por la prematura opacificación de la aorta si la intercomunicación es en sentido inverso.

RESUME

Le diagnostic d'un défaut de la paroi ventriculaire sans complication est fondé sur la présence d'un gros frémissement systolique, et d'un souffle systolique prolongé, et bruyant maximum entre la quatrième et le cinquième espaces parasternaux gauches. Ces signes sont trouvés chez un individu sans cyanose, chez qui l'on constate une hypertrophie ventriculaire gauche, et une saillie de l'artère pulmonaire avec élargissement de l'aorte. Le cathétérisme cardiaque permet de se renseigner valablement sur l'importance du shunt, la surcharge cardiaque, et l'état hémodynamique du lit vasculaire pulmonaire. Les procédés angiocardiographiques aident au diagnostic en mettant en évidence le trouble de remplissage du ventricule droit pendant l'opacification du coeur droit ou l'opacification prématurée de l'aorte si le shunt est en sens contraire.

ZUSAMMENFASSUNG

Die Diagnose eines nicht komplizierten Veutrikel-Septum Defektes hat zur Grundlage das Vorliegen eines rauhen systolischen durchringenden und langen, harten systolischen Geräusches mit maximum im 4. und 5. linken parasternalen Intercostalraum bei einem nicht cyanotischen Individuum, bei dem sich Anhaltspunkte finden für eine Hypertrophie des linken Ventrikels und für Vorstehen der Pulmonal-Arterie ohne Vergrosserung der Aorta. Herzkatheterisierung ergibt einen wertvollen Nachweis über die Grösse des Kurzschlusses, die Herzbelastung und die haemodynamische Verfassung des Lungengefässsystems. Angiocardiographische Untersuchungen unterstützen die Diagnosestellung durch Nachweis eines Füllungsdefektes im rechten Ventrikel während der Kontrastfüllung des

rechten Herzens oder durch eine vorzeitige Konstrastfüllung der Aorta, falls der Kurzschluss entgegengestzt ist.

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Circulatory and Pulmonary Effects of Acute Central Vascular Engorgement in Normal Subjects*,**

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Chronic pulmonary vascular engorgement has been extensively studied in patients with congestive heart failure. It is frequently difficult under these conditions to establish with precision those features which are due solely to central vascular engorgement and those which are related to the underlying disease.

To investigate the respiratory and circulatory effects of acute pulmonary vascular engorgement in normal subjects, observations have been made during inflation of an aviators g-suit and submersion in water.

Inflation of a single chamber g-suit results in rapid application of pressure to an area of the body corresponding to that covered by trousers. The blood is forced centrally. During and after g-suit inflation continuous measurements were made of tidal volume and intraesophageal pressure change. Liters of respiratory volume change per centimeter of water intrathoracic pressure change is a reciprocal index of the stiffness of the lung, the pulmonary compliance. The stiffer the lung the lower the compliance.

In addition, central venous pressure was measured by a standard technique and pulmonary vascular density was followed by x-ray as the g-suit was rapidly inflated and maintained at a pressure of 2 psi for 2 minutes.

Chart I summarizes the data obtained in a series of 15 subjects studied in the seated position. As on subsequent charts, each value presented here represents the group mean. Individual values used in computing the mean compliance were in turn the mean of five determinations.

Within 30 seconds after suit inflation compliance decreased from a mean of .217 to .093 L/cm. H_2O. Central venous pressure increased by 274 mm. H_2O concomitant with a marked increased in radiographic pulmonary vascular density. These are indications of central and pulmonary vascular engorgement which produced, under the acute experimental conditions compliance change quantitatively comparable to that of congestive heart failure. It is of interest that there was no change in respiratory resistance during g-suit inflation.

Throughout the two minutes of constant g-suit pressure, the lungs became less stiff as reflected by the increase in compliance. Central venous pressure decreased progressively. Concomitantly there was a marked decrease in pulmonary vascular density. The changes in compliance and in central venous pressure during the period of constant g-suit pressure are significant at the $p<.001$ level.

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CHART I
EFFECT OF CONSTANT G SUIT PRESSURE (2 psi) ON PULMONARY COMPLIANCE AND CENTRAL VENOUS PRESSURE (15 Subjects)

	Time After Suit Inflation			
	Control	30 Sec.	60 Sec.	120 Sec.
Pulmonary compliance (L/cm. H_2O)	.217 ± .048	.093 ± .036	.114 ± .027	.141 ± .046
Central venous pressure (mm. H_2O)	13 ± 4	274 ± 75	230 ± 74	195 ± 63

These findings suggested that the initial central vascular engorgement was followed by a decrease in central blood volume during the period of constant g-suit pressure.

To evaluate the effect of a more even application of pressure over the entire body, pulmonary compliance was measured as eight seated subjects were submerged in water while breathing against atmospheric pressure.

Chart II presents a summary of this data. Pulmonary compliance decreased within 30 seconds to .099 with submersion to the neck, and to .077 with total submersion. During two minutes submersion, compliance increased significantly in each instance.

In both series of experiments, it seemed that the initial central vascular engorgement was followed by a redistribution of blood effecting a decrease in central blood volume despite constant pressure on the surface of the body.

To elucidate the factors responsible for this centrifugal redistribution of blood, subjects were observed during g-suit inflation while receiving drugs which modify vascular tone.

Four subjects received intravenous hexamethonium sufficient to lower arterial pressure to a mean of 65/45. G-suit inflation at this time resulted in a less marked increase in central venous pressure although the fall in central venous pressure with constant g-suit pressure was again apparent.

Pulmonary compliance decreased with suit inflation and, after two minutes, had again increased significantly. Partial ganglionic blockade with hexamethonium lessens the increase in central venous pressure with g-suit inflation but does not alter subsequent apparent redistribution of blood.

Five subjects were studied during the infusion of nor-epinephrine sufficient to raise arterial pressure to a group mean of 180/100.

As indicated in Chart III, nor-epinephrine infusion *per se* produced an increase in central venous pressure and a decrease in compliance. G-suit

CHART II
EFFECT OF IMMERSION ON PULMONARY COMPLIANCE

	Pulmonary Compliance		
	30 Seconds Immersion	120 Seconds Immersion	Significance of Change—P
Immersion to neck	.099 ± 0.27	.116 ± .039	.05
Total immersion	.077 ± .027	.100 ± .021	.01

inflation at this time resulted in a marked increase in central venous pressure to 401 mm. H_2O and a decrease in compliance to .064. Subsequently there was no significant change in central venous pressure or pulmonary compliance during the two minutes of suit inflation. Two subjects were studied during g-suit inflation while one arm was immersed in ice water. As with the nor-epinephrine infusion, there was no significant change in central venous pressure or pulmonary compliance during the two minutes of constant g-suit pressure.

The centrifugal shift of blood during central vascular engorgement is not seen during norepinephrine infusion or immersion of one arm in ice water. This redistribution of blood seems related to peripheral vascular tone. Because of the volume change involved it appears probable that venous tone plays a significant role in this respect.

In summary, diffuse pressure over the surface of the body results in an increase in central venous pressure and radiographic pulmonary vascular density. Concomitantly the lungs become more stiff as manifest by a decreased compliance. These are reflections of central vascular engorgement. As pressure is maintained on the surface of the body, decreasing central venous pressure and raiographic pulmonary vascular density with increasing pulmonary compliance all suggest a shift of blood to the periphery.

Nor-epinephrine infusion or immersion of an arm in ice water prevents this redistribution of blood during central vascular engorgement. It would seem therefore that the ability of the normal subject to shift blood out of the central vasculature during central engorgement is related to peripheral vascular tone.

SUMMARY

1. Brief, acute central vascular and pulmonary engorgement has been produced in normal subjects by inflation of an aviator's g-suit and by submersion in water while breathing against atmospheric pressure.

2. G-suit inflation (2 psi) produced an increase in central venous pressure of 26.8-7.8 cm H_2O and a decrease in pulmonary compliance from .217-.048 to .093-.136 L per cm H_2O. A similar compliance change occurred with submersion.

CHART III

EFFECT OF NOR-EPINEPHRINE INFUSION ON PULMONARY COMPLIANCE AND CENTRAL VENOUS PRESSURE DURING G-SUIT INFLATION (2 psi) (5 Subjects)

	Time After Suit Inflation			
Control	Control	30 Sec.	60 Sec.	120 Sec.
Pulmonary compliance (L/cm. H_2O)	.204 ± .013	.089 ± .022	.104 ± .023	.114 ± .028
Central venous pressure (mm. H_2O)	13 ± 6	259 ± 64	219 ± 50	183 ± 61
Nor-epinephrine infusion 0.4-0.6 mg./Kg./min.				
Pulmonary compliance (L/cm. H_2O)	.162 ± .036	.064 ± .015	.066 ± .015	.068 ± .015
Central venous pressure (mm. H_2O)	65 ± 20	401 ± 55	405 ± 67	408 ± 70

3. On maintaining g-suit pressure or submersion for two minutes, central venous pressure and compliance showed a progressive but incomplete return toward control levels.

4. Infusion of norepinephrine or immersion of an arm in ice water during g-suit inflation prevented the return of central venous pressure and pulmonary compliance toward control levels.

5. It is suggested that the compliance changes induced by these procedures depend partially on the degree of central vascular engorgement and that under the experimental conditions this in turn is partially dependent on peripheral venous tone.

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RESUMEN

1. Se produjo una ingurgitación aguda vascular central y pulmonar breve en sujetos normales cuando se infla envestido "G" especial de aviadores y son sumergidos en agua al mismo tiempo que respiran aire a presión atmosférica.

2. El inflado de los vestidos "G" (2 psi) produce un aumento de la presión venosa central de 26.8-7.8 cms. H_2O y un aumento en al rendimiento pulmonar de .217-.048 a .093-.136 L por cm. H_2O. Un cambio semejante en rendimiento ocurrió por la sumersión.

3. Manteniendo un vestido "G" a presión o la sumersión por dos minutos la presión central venosa y el rendimiento mostraron un retorno progresivo pero incompleto hacia los niveles normales.

4. La infusión de norepinefrina o la inmersión de un brazo en agua helada durante el inflado del vestido "G" evitó el regreso de la presión venosa central y el rendimiento pulmonar hacia lo normal.

5. Se sugiere que los cambios de rendimiento producidos por estos procedimientos dependen en parte de ingurgitación vascular central y que bajo las condiciones experimentales, esto a su vez depende parcialmente del tono venos periférico.

RESUME

1. Un engorgement pulmonaire et vasculaire bref et aigu a été produit chez des individus normaux par gonflement de combinaisons d'aviateur et immersion dans l'eau tandis que la respiration s'effectuait contre la pression atmosphérique.

2. L'augmentation de la pression de l'air de la combinaison produit une augmentation de la pression veineuse centrale de 26,8 au lieu de 7,8 cm. d'eau et une diminution des besoins pulmonaires. Une altération semblable des besoins survient avec l'immersion.

3. En maintenant la pression de la combinaison ou l'immersion pendant deux minutes, la pression veineuse centrale et les besoins montrent un retour progressif mais incomplet aux taux témoins.

4. L'injection de norepinephrine, ou l'immersion d'un bras dans l'eau glacée pendant le gonflement de la combinaison empêcha le retour de la pression veineuse centrale et des besoins pulmonaires aux niveaux normaux.

5. L'auteur pense que les changements dans les besoins produits par ces moyens dépendent partiellement du degré de l'engorgement vasculaire central, et que dans les conditions d'experimentation ceci à son tour est partiellement dépendant du tonus veineux périphérique.

ZUSAMMENFASSUNG

1. Eine Kurze, akute, zentrale vasculäre und pulmonale Hyperaemie wurde bei normalen Personen erreicht, durch Aufpumpen einer Fliegergarnitur g und Untertauchen im Wasser bei gleichzeitiger Atmung gegen atmosphärischen Druck.

2. Das Aufpumpen dieser Garnitur (2 psi) führte zu einer Erhöhung im zentralen Venendruck von 26,8-7,8 cm. H_2O und einer Abnahme der Lungenfüllung von .217-.048 auf .093-136 L per cm. H_2O. Eine ähnliche Abnahme trat beim Untertauchen ein.

3. Wurde der Druck in der Garnitur p oder das Untertauchen 2 Minuten lang durchgehalten, zeigten zentraler Venendruck und Füllung eine schrittweise, jedoch unvollständige Rückkehr zu den Kontrollwerten.

4. Infusion von Norepinephrin oder Eintauchen eines Armes in Eiswasser während dem Aufblasen der Garnitur p hemmte den Rückgang des zentralen Venendrucks und der pulmonalen Füllung auf die Kontrollwerte.

5. Wird angenommen, dass die durch diese Massnahmen herbeigeführten Änderungen in der Füllung zum Teil abhängig sind vom Grad der zentralen vasculären Hyperaemie und das unter den Versuchsbedingungen diese ihrerseits zum Teil abhängt vom peripheren Venentonus.

Coronary Curettage

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Despite the number one etiologic position of occlusive heart disease in the hierarchy of per annum death rates, an energetic approach to the problem is conspiciously absent. Physicians are more or less content with supportive treatment and mildly encouraging medical adjuncts, chief among which are production of hypothyroidism by radioactive iodine and other methods,[1] low cholesterol diets,[2a, b] feminizing hormones,[3a, b, c, d] anticoagulants,[4a, b, c, d] and parenteral heparin for its lipotropic effect.[5a, b] In recent years, for more startling and dramatic improvement in those already afflicted, one has looked to the cardiac surgeon, and will probably continue to do so, until the real solution to the problem of coronary heart disease is found in the realm of prophylaxis.

Attempts to revascularize and/or improve collateral circulation to the heart have been undertaken with ingenuity and persistence. Beck returned to his original concept of enhancing myocardial perfusion by poudrage,[16] having abandoned the more formidable aortic coronary sinus anastomosis.[7] Louis Gross sought to improve coronary collateral circulation by partial occlusion of the coronary sinus, reasoning that the increased venous head pressure would stimulate multiplication of arterial labyrinths.[8a, b] The implantation of the internal mammary artery into the myocardium as advocated by Vineberg[9a, b] is purported to establish direct communications between budding branches of the implanted vessel with coronary branches. The attachment of omentum,[10] lung,[11] pectoralis muscle,[6] diaphragm[12] or intestine[13] to the myocardium have likewise been attempted, each with varying claims for improvement. This much may be said for most of the above enumerated indirect approaches: Experimental studies indicate that by them blood flow is increased via collateral circulation by 1-4 cms/min.[14a, b] and that this increment is by and large sufficient to reduce ligation deaths and size of infarcts in the experimental animal.[14c] Since, all things being equal, the simplest method is most desirable, the insertion of asbestos,[15, 15a] or U.S.P. talc[16] into the pericardium are the methods generally employed; clinical improvement is claimed for 60-80 per cent of patients with coronary artery disease and angina. The improvement is said to reflect in diminishing need for nitroglycerin and increased work capacity.

More recently, Angelo May has suggested a direct approach.[17, 20] In the experimental animal he was able to traverse coronary arteries with a specially constructed needle-probe whose cutting under surface on the lead heads function on withdrawal. On this basis he urged the clinical "reaming out" of the coronary arteries. Putting May's method to test, Bailey reported its successful application in two patients,[18] and subsequently added three more cases with promising clinical results.[19] All his

Preclinical experimental work was supported by the Department of Research, Temple Hospital, Los Angeles, and Research Grant from National Heart Institute, Bethesda, Maryland.

cases had marked angina with a history of previous coronary artery occlusion. His oldest patient was 54 years of age. A sixth case was added by the authors on March 20, 1957. The outstanding features in this case were the fact that the patient had a previous poudrage in December 1955 which failed to alleviate symptoms, that he was 58 years old, thereby extending the age limit of the procedure, that a flexible probe replaced the rigid model initially developed by May, and that dilatation of the vessels was added to, and considered an important part of the method.

Case Report: A 58 year old white man was admitted to Temple Hospital, Los Angeles, on March 11, 1957 with the chief complaint of severe angina. In 1939 he had a posterior myocardial infarction for which he was hospitalized for four weeks. Since then he had several hospitalizations because of incapacitating angina pectoris, and on one occasion for an episode of hypertension. The angina became progressively worse, occurring after walking a few steps, after meals, and in cold weather at rest. Pericardial poudrage was undertaken elsewhere in December 1955. Improvement followed until resumption of full activity, whereupon angina recurred with increasing intensity. For six months prior to the last hospitalization he was confined to his home, mainly in bed, and in addition to usual medication for angina had oxygen on hand for frequent use. Nitroglycerin gave short relief and was taken before meals to prevent post cibum angina.

Pertinent past history featured gastric resection in 1947 for peptic ulcer, followed by vagotomy in 1948.

Family history: Father died at 66 of "heart attack." Mother "dropped dead" at 64. Two brothers are living, both with coronary artery disease. Two sisters are living, one has coronary artery disease. Pertinent physical findings on admission: Temperature 98.6°F. Blood Pressure 130/80. Pulse Rate 80. "Patient is a ruddy faced, slightly overweight white man.

There is bilateral arcus senilis and full dentures. There is no neck vein engorgement on recumbency. Chest reveals a well healed anterior horizontal scar, over the left fifth cartilage from the previous poudrage operation. There is definite thoracic emphysema. Percussion of chest reveals the hyper-resonance of pulmonary emphysema except for dullness at the left posterior base. Breath sounds are distant; on forced expiration, scattered, prolonged wheezes are audible. The heart is not enlarged. A_2 less than P_2. A grade I systolic murmur is present over the mitral valve area. Abdomen

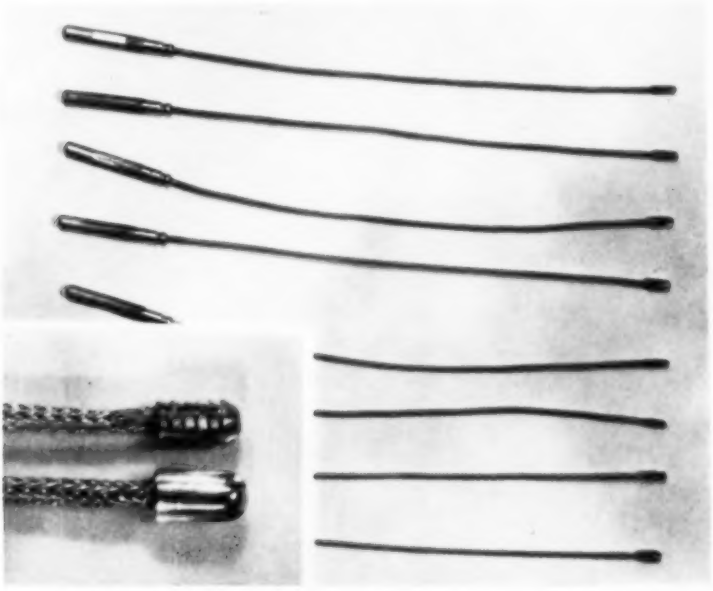

FIGURE 1: Graduated flexible coronary artery curettes and probes.

is obese with no palpable viscera. Upper abdominal scars of previous surgery are well healed. Extremities are normal. Brachial vessels are sclerotic but brachial and radial pulses are strong. Femoral and popliteal pulses are present, dorsalis pedis and posterior tibials are barely palpable."

Pertinent laboratory findings were: Complete blood count, urinalysis, fasting blood sugar, and blood urea nitrogen were within normal limits. Serum cholesterol was 250 mm. per cent (normal 180-220). Chest x-ray film revealed a normal cardiovascular shadow. There was pulmonary emphysema and thickening of the pleura at the left base. Preoperative ECG's revealed evidence of an old posterior myocardial infarction and an antero-lateral myocardial ischemia pattern. T_1, T_{AVL}, and T-$V_{5, 6}$ were inverted. There was slight depression of $ST_{2, 3}$ and AVF. A Q_{AVL} (Figure 3) was also present. Review of ECG's taken on March 23, 1953 and December 23, 1955 revealed similar patterns with deeper Q_{AVL} and taller left precordial R waves. The patient suffered attacks of angina while hospitalized at complete bed rest; a prolonged attack occurred on the morning of surgery.

On March 20, 1957 under Epidural anesthesia, with light pentothal, coronary curettage was carried out as follows:

The left hemithorax was entered through the bed of the sixth rib, and excellent exposure provided by means of a Finochietto rib spreader. Immediate inspection revealed a highly organized thick fibrothorax involving the lower left lung and adherent to the pericardium as well as the thoracic wall and diaphragm. Decortication was performed and the lung completely mobilized. The pericardium was thickened. Tortuous, thickened coronary vessels were discernible by palpation. The pericardium was incised medial to the phrenic nerve, along the entire length of the left ventricle. A few adhesions anteriorly were easily separated, and the epicardium was freed from the pericardium posteriorly by blunt finger dissection; in only one or two places were the adhesions firm enough to require cutting with scissors. The left anterior descending coronary artery was visualized and found to be markedly sclerotic and tortuous; two-thirds distant from its origin, its course followed an acute S-shape. Terminally it disappeared into the myocardium before reaching the apex.

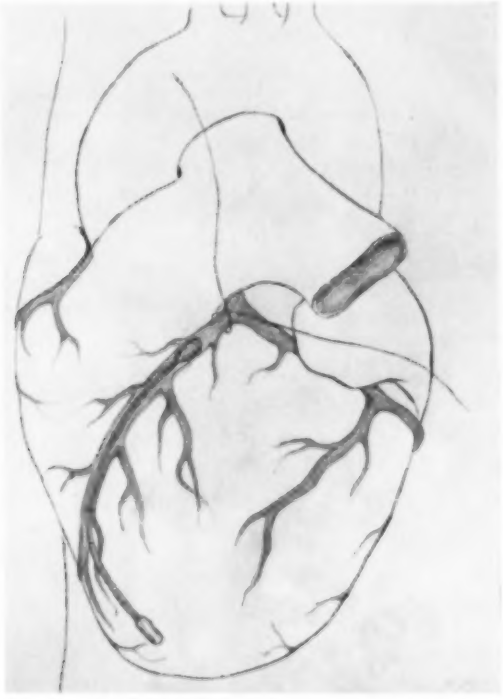

FIGURE 2: Diagram of operative technique. Note hemostatic control suture beneath proximal anterior descending coronary artery. A curette is shown passing up lumen of anterior descending coronary artery.

The edges of the pericardium were held apart by means of traction sutures. A No. 000 cotton atraumatic suture was placed under the origin of the anterior descending coronary artery for subsequent hemostasis. On crossing the free ends to test occlusion of the vessel, a few premature contractions of the ventricle were noted. These disappeared spontaneously, even before releasing tension on the suture. No. 00000 atraumatic silk sutures were placed in the epicardium overlying the terminal portion of the left anterior descending coronary artery. The epicardium was freed and an incision was made into the coronary artery terminally. It bled slightly; this was controlled by crossing the ends of the previously placed hemostatic suture. A No. 2 flexible coronary artery curette (Figure 1)* was inserted in the coronary artery and directed upward (Figure 2). It passed easily until it reached the S-shaped portion of the artery where it could not make the turn without hazarding perforation of the vessel. It was withdrawn and followed by the insertion of the No. 2 coronary artery dilator. Following this maneuver, it was apparent that blood flow was decidedly increased. Two more attempts were made to pass the probe beyond the S-curve in the vessel; these attempts were abandoned in view of the possible dangers. The table was rotated toward the operator to present a better view of the posterior portion of the left ventricle. The pericardium was retracted laterally, exposing the lateral branch of the circumflex artery which was also found to be sclerotic and tortuous. Two stay sutures of No. 00000 braided atraumatic silk were placed on either side of the terminal portion of this vessel, grasping the epicardium. A longitudinal incision was made in the

*The graduated Satinsky coronary artery curettes and dilators may be obtained from George P. Pilling & Son Co., Philadelphia, Pa.

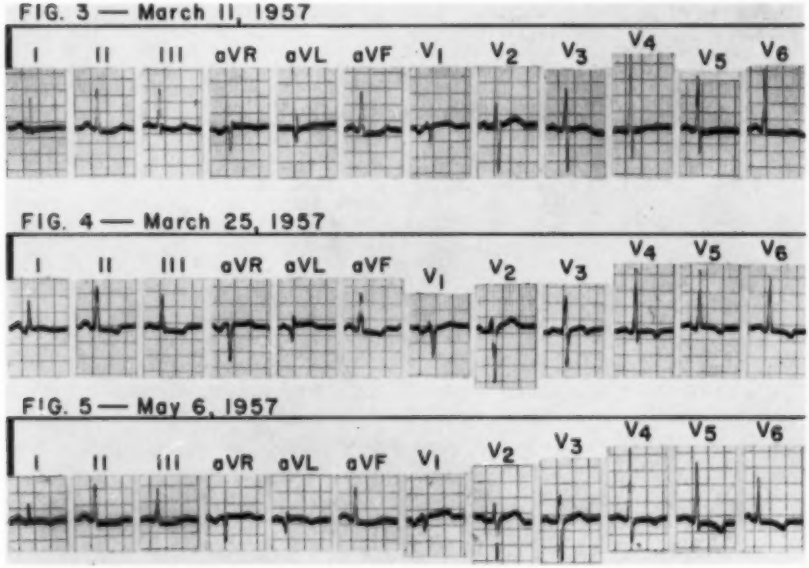
FIG. 3 — March 11, 1957
I II III aVR aVL aVF V1 V2 V3 V4 V5 V6
FIG. 4 — March 25, 1957
I II III aVR aVL aVF V1 V2 V3 V4 V5 V6
FIG. 5 — May 6, 1957
I II III aVR aVL aVF V1 V2 V3 V4 V5 V6

Figure 3: Pre-operative ECG revealing evidence of old posterior myocardial infarction and antero-lateral ischemia.—*Figure 4:* ECG on fourth post-operative day. Antero-septal anoxia is increased. T AVR now reciprocally upright.—*Figure 5:* ECG six weeks post-operatively, similar to pre-operative ECG except for deeper T wave inversion in respective leads; significantly, there was no QRS change at any time.

FIGURE 6: ECG 12 weeks post-operatively shows progressive improvement.

terminal portion of the vessel and the No. 2 flexible coronary artery curette was inserted into the lumen of the artery. It passed easily through the entire length of the vessel directly into the circumflex artery. Following its removal, blood flow increased. Small pieces of tissue came away with the curette. This maneuver was followed by the insertion of the corresponding coronary artery dilator. This too passed through the vessel with remarkable ease. Therefore, the No. 3 curette and dilator were inserted into the vessel with further improvement in blood flow. The open end of the bleeding vessel was controlled by oversewing the epicardium. Meanwhile, blood flow likewise increased in the region of the anterior descending coronary artery which had been incised: A definite "pumper" was visualized in contradistinction to the absence of pulsatile flow preoperatively. This was controlled with an epicardial 5-0 atraumatic suture.

Throughout surgery the electrocardiogram was monitored continuously by cardioscope. At no time did the heart show any significant changes during the above maneuvers with the exception of transient premature extasystoles when the proximal portion of the anterior descending coronary artery was electively occluded for purposes of hemostasis.

The pericardium was closed to its apex where a small window was left for drainage. The hemithorax was irrigated and the lung re-expanded. An intercostal tube was placed at the lowermost portion of the left hemithorax for drainage and connected to a waterseal system of bottles, and the left chest was reconstituted in layers.

The post-operative course was surprisingly free of complications. On the third post-operative day, the patient was anti-coagulated with heparin, but this was discontinued because of hemoptysis presumed to be due to the extensive decortication of the left lung. There were mild asthmatic rales and rhonchi with expectoration of purulent sputum the first post-operative week. Serial daily Transaminase (SGO-T) values the first, second and third post-operative days were 65, 63, and 55 units.

On March 19, 1957, the first post-operative day, the ECG revealed a mild injury pattern over the right precordial leads consisting of slight ST elevation in lead I and V_{1-3}. T_1 was more inverted, T_2 was now inverted, T_{AVR} was now upright. By March 25, 1957, the ECG had stabilized and except for very minor T wave changes remained the same throughout hospitalization, the deeper T wave inversion in leads 1, 2, AVF, $V_{3,4,5}$ being noteworthy (Figure 4).

The patient was permitted out of bed on the 10th post-operative day. He was surprised to discover that he could walk up and down the hall without angina. He was discharged April 3, 1957, 13 days after surgery, and allowed gradual activity. On May 6, 1957, six weeks after surgery, he drove his car approximately 200 miles for his checkup; he walked up a hill for a distance of 100 yards at a rise of 70 feet. His only complaint was a little fatigue. He had no angina. ECG now resembled the pre-operative ECG's except for deeper inversion of T waves in leads 1, AVL, V_{1-3} (Figure 5). Significantly, since surgery there have been no QRS changes.

SUMMARY AND CONCLUSION

A case of coronary artery curettage on a 58 year old white man with intractable angina upon whom a poudrage was previously performed is reported. The spectacular improvement in this case, together with the beneficial results in Bailey's five cases suggest that this procedure deserves the further attention of clinician and investigator.

ADDENDUM: Patient was last seen September 1958, 18 months after coronary curettage. He is actively engaged in the same occupation, without restriction. Physical examination, cardiac x-rays and fluoroscopy, and ECG findings have not changed.

RESUMEN

Se relata un caso de un hombre blanco de 58 años en el que se hizo raspa de la arteria coronaria para una angina de pecho intratable que ya había sido antes sujeta a tratamiento por polvo intrapericárdico.

La mejoría espectacular en este caso, además del benéfico resultado obtenido en los cinco casos de Bayley, sugiere que este procedimiento merece mayor atención de los clínicos y de los investigadores.

RESUME

L'auteur rapporte un cas de curetage de l'artère coronaire chez un homme âgé de 58 ans, de race blanche, atteint d'angine de poitrine incu-

rable, et sur lequel on avait déjà pratiqué antérieurement un talcage. L'amélioration spectaculaire dans ce cas, ainsi que le résultat favorable des cinq cas de Bailey, amène à penser que ce prodédé légitime l'attention du médecin et du chercheur.

ZUSAMMENFASSUNG

Bericht über einen Fall einer Ausschabung der Koronararterie bei einem 58 Jahre alten Weissen mit unbeeinflussbarer Angina, bei dem zuvor eine Puderbehandlung vorgenommen war. Die auffällige Besserung dieses Falles zusammen mit dem günstigen Resultat in Bailey's 5 Fällen deutet darauf hin, dass dieses Verfahren weitere Aufmerksamkeit von Klinikern und Forschern verdient.

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Reasons for the Unsettled State of the Anticoagulant Controversy

Still unsettled is the controversy over the use of anticoagulants in acute and chronic coronary heart disease some 15 years after the first reports appeared. A consideration of the reasons that prevent the solution of this vexing problem may be of more value than participating in a fruitless debate.

1. We are dealing with a disease—acute myocardial infarction— of unknown primary cause. Ignorance of the causation of a malady nearly always leads to faulty treatment that is modified or discarded as knowledge is accumulated. The reduction of strict bed-rest from three months to three weeks or less (with toilet privileges included) in this disease should make us pause before we take sides in the anticoagulant controversy. An hour with a half-century old text-book of medicine should be convincing.

2. Many cases of acute myocardial infarction are not strikingly similar in the clinical sense, as are, for example, most cases of subacute bacterial endocarditis. In the Framingham long-term epidemiologic study,[1] one-fourth of the coronary attacks were found to be silent. Electrocardiograms revealed acute myocardial infarction without symptoms in 9 per cent, and with vague symptoms, erroneously interpreted by both patients and doctors, in 16 per cent. As the clinician has learned, one coronary patient has mild pain and is alert, peppy and cheerful, while another is sick with recurrent pain, clammy skin and tachycardia. Such a disease of unknown cause wherein one case often is hardly like the next plays havoc with a series of alternate patients, and makes less reliable any system of assessing good-risk and poor-risk cases by criteria often inexact, like prior myocardial infarction.

3. Many cases of acute myocardial infarction are not directly caused by acute coronary thrombosis. The clinician assumes that a thrombus in a coronary artery initiates the myocardial infarction he is called upon to treat. He administers anticoagulants, among other reasons, to prevent extension of the thrombus. However, the incidence at autopsy of thrombosis as a cause of myocardial infarction varies from 21 per cent to 95 per cent, as shown in Table I.

At best, one can draw a rough average and agree with Yater[3] that "death from coronary artery disease is associated with coronary thrombosis in about 50 per cent of the cases." We clinicians must keep this 50 per cent figure in mind when we administer anticoagulants to prevent extension of a thrombus that might be non-existent.

4. The anatomical distribution of the coronary arteries (preponderant right or left, or balanced) is an indeterminate factor influencing the immediate mortality and future life-span of the patient with acute myocardial infarction.[4] The invisible coronary topography makes of alternate cases and matched series a hodge-podge of unpredictability. This is

TABLE I
INCIDENCE OF THROMBOSIS AS THE CAUSE OF CORONARY OCCLUSION
[modified from Bjerkelund (2)]

Investigator	No. Cases	Cases With Thrombosis	Per Cent With Thrombosis
Wearn (1923)	19	18	95
Nathanson (1925)	113	24	21
Parkinson & Bedford (1928)	40	29	72
Levine & Brown (1929)	46	23	50
Lisa & Ring (1932)	32	13	41
Applebaum & Nicolson (1935)	150	78	52
Saphir et al. (1935)	34	25	74
Bean (1938)	300	240	80
Horn & Finkelstein (1940)	172	136	79
French & Dock (1944)	80	29	36
Yater et al. (1948)	450	225	50
Papacharalampous & Zollinger (1953)	126	78	62
Wright, Marple & Beck (1954)	89	64	72

demonstrated in the paragraph that follows.

5. The mortality rate and the survival time after sustaining acute myocardial infarction are not predictable numbers, a reflection of the insoluble problems stated in the previous paragraphs. The immediate mortality rate varies from 3.1 per cent to 51.5 per cent. The chances to be alive three years after a heart attack varies from 41 per cent to 87 per cent; to be alive five years after an attack varies from 22 per cent to 82 per cent.[5] If various series of cases not treated with anticoagulants vary so widely in prognosis because of the many imponderable and unpredictable factors that constitute the disease we classify as acute myocardial infarction, how can the effect of an anticoagulant be trustworthily assayed? Combining several small series merely compounds error upon error.

6. Anticoagulants cannot be expected to forestall death in all cases of acute myocardial infarction. Ventricular fibrillation, asystole and ventricular rupture, the usual causes of sudden death in the first seven to 10 days, cannot be prevented by anticoagulants. Unfortunately, the mechanisms of sudden death are not known.

TABLE 2
CAUSES OF DEATH AFTER HEALED MYOCARDIAL INFARCTION
[constructed from data quoted by Bjerkelund (2)]

	No. Cases	New Infarction	Sudden Death	Heart Failure	Non-cardiac
Levine & Rosenbaum (1941)	80	40	35	20	5 per cent
Sigler (1951)	393	68 per cent	16 per cent	15 per cent	1
Katz (1954)	171	55		17	10

7. Although oral anticoagulants were first used to prevent thromboembolic complications of myocardial infarction, the indications for their use have expanded to include long-term prophylaxis in coronary disease. Few can deny that anticoagulants have nearly halved the appreciable mortality from complicating pulmonary thromboembolism. However, oral anticoagulants have been recommended after surviving acute myocardial infarction by Bjerkelund[2] for only one year and only in young patients; and by Wright[6] for the remainder of life in all patients. Angina pectoris with or without proved cardiac infarction, preinfarction pain, and impending myocardial infarction (is this analogous to impending preg nancy?) are now being treated with anticoagulants. Unquestionably, the post-infarction patient is most likely to die of heart disease (Table II). But only about 50 per cent will die of another acute myocardial infarction, in whom, considering paragraph three above, approximately only half the cases would show coronary thrombosis that could possibly be prevented by anticoagulants. Sudden death is not prevented by long-term anticoagulants, their proponents admit.[7]

6. Dividing the patients into good-risk and poor-risk classes as a guide to the use of anticoagulants is not fool-proof. Except for the obvious danger-signs of severe shock, ventricular arrhythmias and acute pulmonary edema, there is open disagreement in the literature concerning the seriousness of increased age, sex, obesity, diabetes, previous angina, previous myocardial infarct, and hypertension. The reader is invited to study of Table III of Eastman et al.[8] for eye-opening data.

7. That anticoagulant drugs can cause infrequent dangerous hemorrhage is not a pertinent argument in this controversy. To be alive is to be in danger; to be sick is to be in double peril.

8. Finally, are the oral anticoagulants antithrombotic agents? The nub of the problem is the prevention of coronary thrombosis and phlebothrombosis, and, hopefully, the prevention of coronary atherosclerosis. An antithrombotic agent is wanted. There is little need to emphasize that oral anticoagulants prevent in most instances the accumulation of a frangible plume of fibrin up the vein on a small venous thrombus. But evidence is accumulating that dicumarol is not an antithrombotic drug. Wessler, Ballon and Katz[9] merit direct quotation: "One of the major pitfalls in the therapy of thromboembolic disease has been the failure to recognize the distinction between the effects of an anticoagulant upon in vitro blood coagulation per se and the in vivo inhibition of thrombosis. These effects need not occur pari passu. That a drug will interfere with the normal coagulation sequence does not provide evidence that it will block thrombosis."

9. A. Carleton Ernstene[10] recently made a telling statement: "The very fact that a question cannot be settled by a large number of investigators studying many patients for a long time suggests that the agent is probably not completely effective. When a therapy appears that is really effective, reasonable investigators can quickly agree on the results."

CONCLUSION

We remain in a quandary. We treat a disease of the coronary arteries of unknown origin with a drug that might not even be antithrombotic. For all we know, what we

today term acute myocardial infarction may turn out to be several distinct diseases, each requiring different treatment.

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ELECTROCARDIOGRAM OF THE MONTH

The author would be pleased to receive comment and controversy from readers in relation to explanations offered.

In the presence of right bundle branch block it is impossible to recognize the presence or absence of electrical effects of right ventricular hypertrophy. Not infrequently the clinical evidence also leaves much doubt.

The electrocardiogram shown here was obtained from a 70 year old woman who suffered moderate dyspnea on exertion. The blood pressure was 170/80 and there was a systolic murmur at the apex. The lungs showed moderate emphysema.

The routine electrocardiogram showed auricular fibrillation and right bundle branch block. Since this conduction defect (right bundle branch block) is common in middle aged and elderly patients in the absence of heart disease no conclusion is warranted regarding the presence of right ventricular hypertrophy that might result from pulmonary disease in this case. Since such evidence might be unmasked by the abolition of the conduction defect by slowing the heart rate the effect of carotid sinus stimulation was recorded.

As may be seen, slowing of the heart rate induced reversion to normal conduction and under these circumstances (beats x, y, z) no evidence of right ventricular hypertrophy can be detected.

This maneuver is occasionally of great value in problems of this kind. Even this occasional value justifies its application to all such problems.

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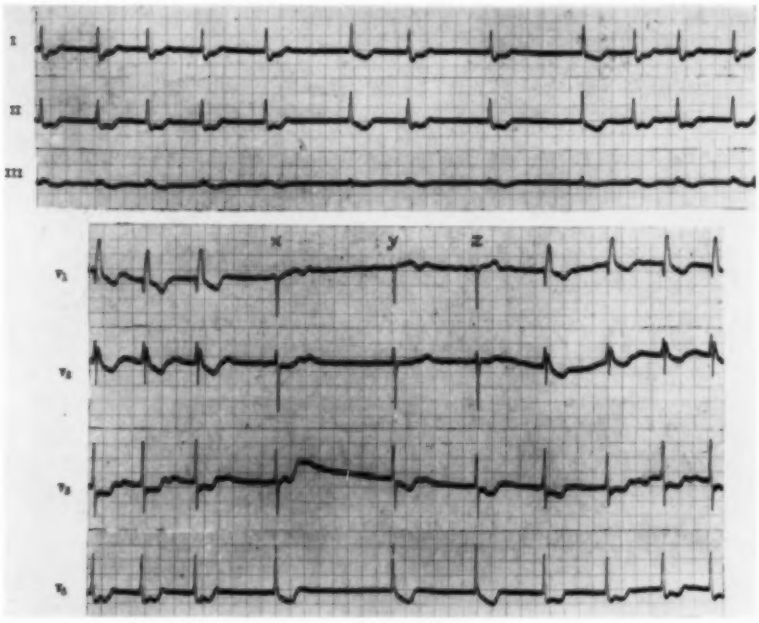
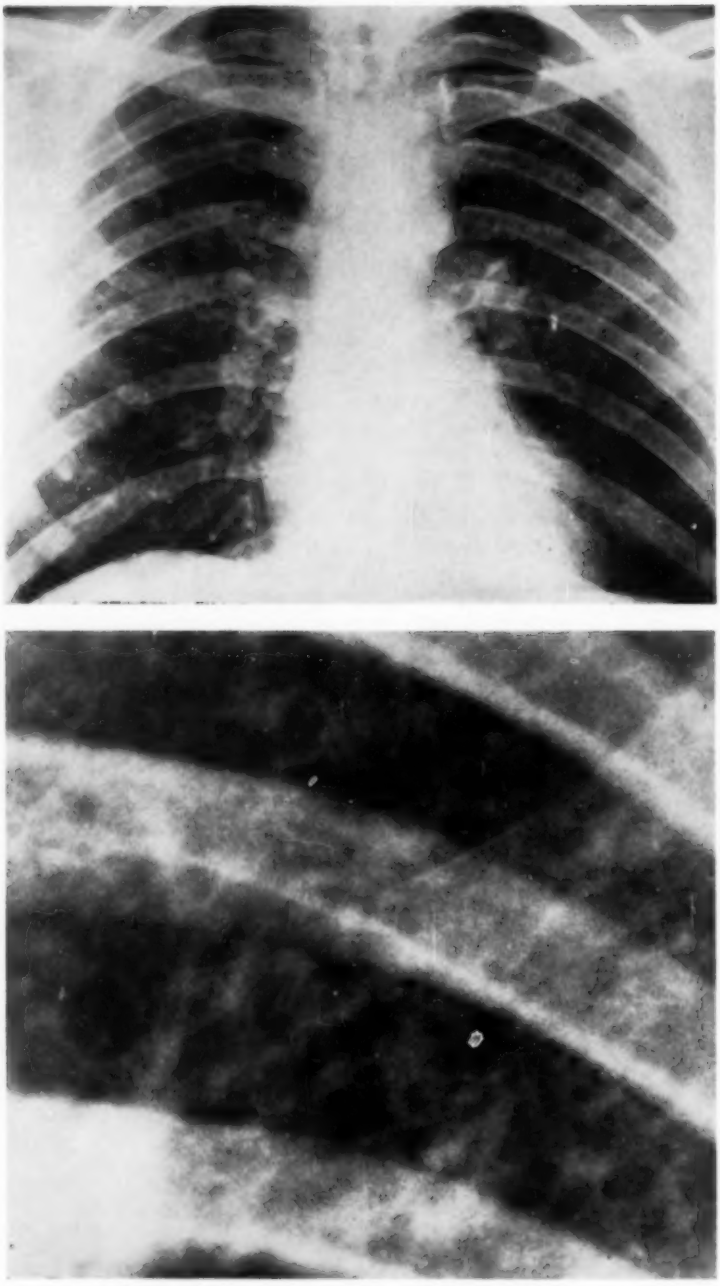


*From Louisiana State University Medical School and Cardiac Research Laboratory, Touro Infirmary.

X-RAY OF THE MONTH



A case of eosinophilic granuloma. For clinical information and diagnosis, see page 101.

Clinical Information

White male, age 28, only symptom was slight productive cough of five months duration, with hemoptysis on two occasions. Except for small non-tender cervical and axillary nodes the physical examination was normal. All laboratory work was normal. Tuberculin and histoplasmin skin tests were positive, complement fixation and agglutination tests for fungus were negative. Sputums were repeatedly negative on smear and culture for tubercle bacilli and fungi.

DIAGNOSIS

Eosinophilic Granuloma of Lung

The lung fields show a diffuse network of fine strands, most prominent in the upper lung field. Throughout are numerous small cystic areas of radiolucency. In some areas miliary nodules are seen. The term *honeycomb lung* has been used to describe this fine cystic pattern. A bone survey was normal.

Thoracotomy with lung biopsy was performed. The lung had a fine cystic appearance and felt nodular. Histologic diagnosis was eosiniphilic granuloma. This diagnosis was confirmed by the Armed Forces Institute of Pathology.

Eosiniphilic granuloma is now widely considered to be closely related to or identical with Hand-Schuller-Christian's disease and Letterer-Siwe's disease. The name *reticulocytosis-X* has been applied to this group of conditions. Eosiniphilic granuloma is the most benign of the three. It may be manifest in the bone as solitary or multiple lesions, in the skin and viscera, and in the lung. The disease may involve multiple systems or affect the lung alone, as seems to have occurred in the present case.

The roentgen findings in the lung are similar in all three of the above conditions. There may be a diffuse reticular network of fine strands accentuated by localized areas of emphysema, often producing the honeycomb appearance. Nodules varying from 2 mm. to 1.5 cm. in diameter with indistinct margins may occur in varying number.

The patient with eosinophilic granuloma may be completely asymptomatic or have a chronic productive cough, weight loss, fatigue, and low grade fever. Rales may be found on physical examination. There are no characteristic laboratory findings. Eosiniphilia is uncommon. Rarely, pleural effusion, spontaneous pneumothorax, or cor pulmonale complicate the usual benign course.

There is no specific treatment, although radiation therapy, steroids and antibiotics have all been tried with varying success. Spontaneous remissions make evaluation difficult.

Honeycombing of the lungs is a relatively uncommon roentgen finding. It may also be encountered in tuberous sclerosis, scleroderma, cystic bronchiectasis, sarcoidosis, and diffuse fibrosis of unknown etiology.

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Case Report Section

Angiosarcoma of the Spleen Complicating Artificial Pneumoperitoneum

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Primary neoplasms of the spleen occur with relative infrequency and of those malignant tumors which have been reported only a few are of vascular origin. In this case the malignancy was associated with an artificial pneumoperitoneum which had been maintained over a long period of time. The exact nature of the abdominal lesion was not apparent until post mortem examination.

Case Report

A 26 year old Negro, was admitted to the Municipal Tuberculosis Sanitarium of Chicago complaining of abdominal swelling and pain, shortness of breath, fatigue, nausea and vomiting. This man had been hospitalized at the Sanitarium seven years before following discovery of a pulmonary lesion, however, after a short stay he left against medical advice to return to his occupation as a bartender. He reported to a private physician at irregular intervals and three years prior to his death pneumoperitoneum was instituted and continued until six weeks prior to his final admission when he began to complain of the abdominal swelling and pain. At this time, he was admitted to a general hospital for a diagnostic workup. Hemorrhagic fluid was removed from the peritoneal cavity and on one occasion acid fast organisms were reported in a smear of the fluid.

Physical examination at the time of admission to the Sanitarium revealed some dullness and cavernous breath sounds over the right lung field. There was marked distension of the abdomen with positive findings for fluid. No abdominal organs could be palpated. A roentgenogram of the chest suggested bilateral apical cavitation.

The total blood serum protein was 4.54 gm. per 100 cc., bilirubin 0.7 mg. per 100 cc., 10 per cent of the bromsulphaline dye was retained and the cephalin flocculation was 3 plus. Cytologic examination of the abdominal fluid revealed some polymorphonuclear leukocytes, a few lymphocytes, masses of red blood cells and on one occasion some cells suggestive of malignancy (Figure 1); the total protein content of the abdominal fluid was 2.5 gm. per 100 cc. Cultures for M. tuberculosis and other microorganisms were negative.

Abdominal paracentesis was performed repeatedly and as much as 2,750 cc. of hemorrhagic fluid were removed at one time. His condition rapidly deteriorated and he expired 10 days after admission to the Sanitarium.

AUTOPSY REPORT

The body was that of a well developed, but poorly nourished Negro 26 years of age and weighing approximately 115 pounds. The abdomen was distended three fingers above the xyphoid process. There were multiple adhesions obliterating the pleural spaces. Along the medial aspect of the left lower lobe there was a friable reddish gray to purple red tumor mass involving the base of the lung and the diaphragm. A small nodule of tumor tissue 3 cm. in diameter was found at the sternal-clavicular junction on the left side. The abdominal viscera were matted together by fibrinous to fibrous adhesions and the left upper quadrant was found to be filled with a friable hemorrhagic reddish gray tumor mass. This involved the spleen, pancreas, stomach, left border of the liver and the diaphragm. Careful dissection revealed that the tumor originated in the hilum of the spleen and invaded the surrounding structures. In addition metastatic foci were present in the liver (Figure 2). Microscopic examination of the tumor revealed a fairly consistent architectural pattern. There were small to large vascular spaces lined by atypical endothelial cells, some of which appeared to be blast forms with large hyperchromatic nuclei (Figures 3 and 4). In other areas there were collections of swollen endothelial cells without any apparent vascular spaces or

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channels. These vascular structures were supported by a stroma which varied from loosely arranged fibroblasts to dense fibrous connective tissue.

DISCUSSION

These malignant tumors of vascular origin have a common pattern but the terminology has been varied and sometimes confusing. After reviewing the literature it is concluded that angiosarcoma would be the most appropriate designation for lesions of this type. Bostik[1] listed seven primary splenic neoplasms based on the cell types found in that organ and malignant vascular lesions were considered to be one of the seven types.

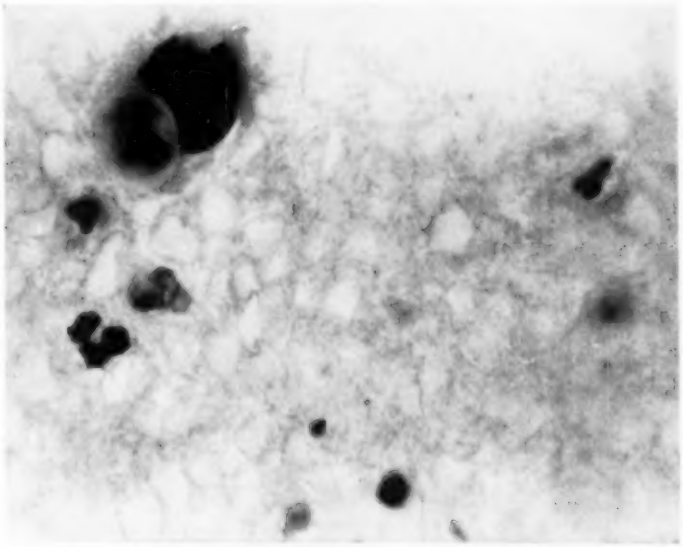

FIG. 1

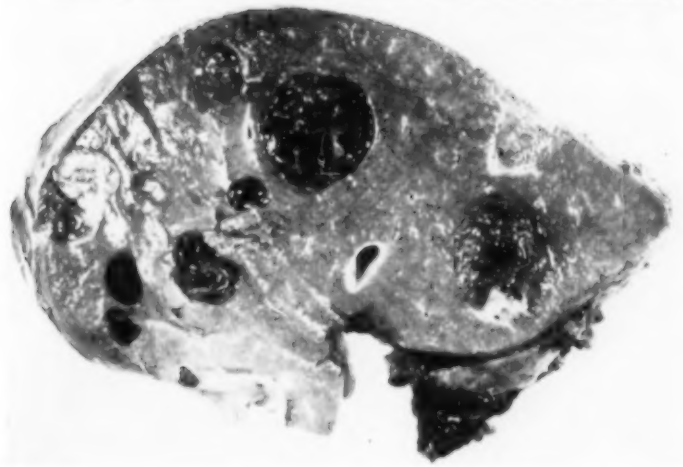

FIG. 2

Figure 1: Smear of abdominal fluid. Note the large atypical cell in the upper left corner (× 900).—*Figure 2:* Gross photograph of the cut surface of the liver. Most of the metastatic lesions are very dark and hemorrhagic but a sub-capsular lesion on the left is pale gray and appears firm.

Regardless of the primary site angiosarcomas show a similar microscopic picture.[3-6] The endothelial cells vary considerably in size and contain large irregular deep staining nuclei. Many of the cells appear to be angioblastic or endothelioblastic. In some instances these endothelial cells are grouped together without any recognizable blood vessel or blood containing space present but in other areas there is vessel budding and in still other areas fully formed blood vessels containing red

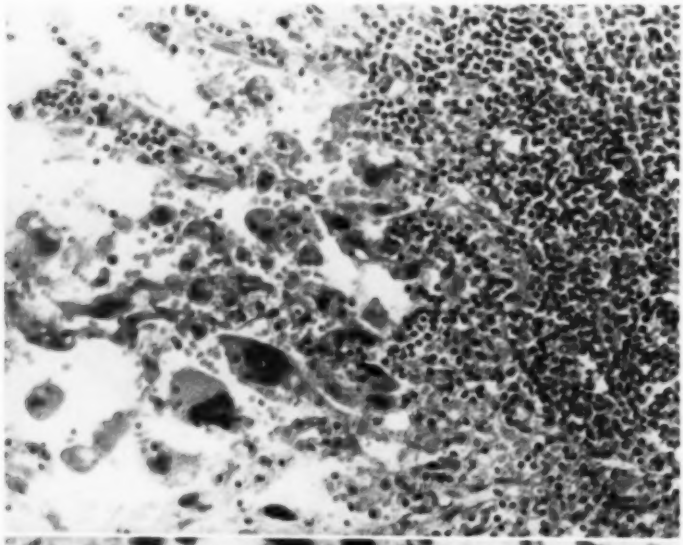

FIG. 3

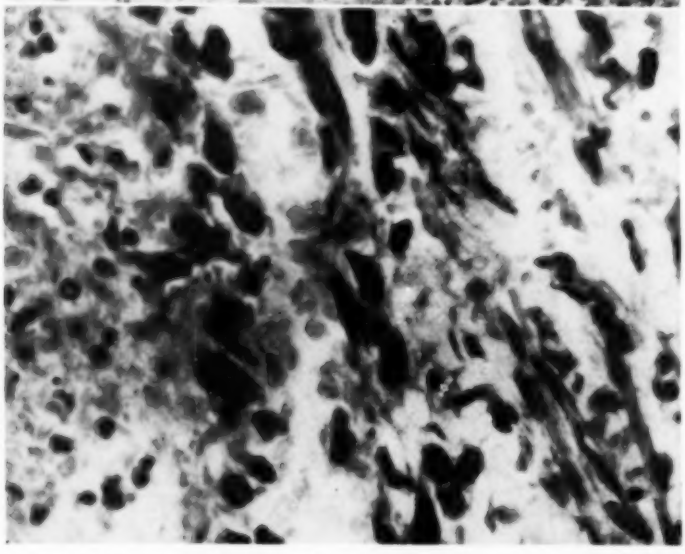

FIG. 4

Figure 3: Photomicrograph showing replacement of splenic parenchyma by tumor. The vascular channels are lined by atypical swollen endothelial cel.s (× 200).—*Figure 4:* A section of the neoplasm showing vascular spaces lined by atypical endothelium. Note the delicate fibrous strands comprising the stroma (× 300).

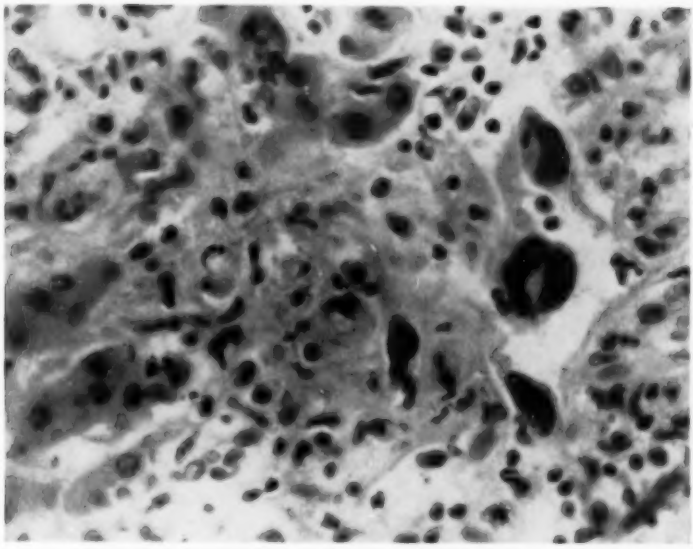

FIGURE 5: In this section of the liver there is an isolated space lined by swollen endothelial cells. This was removed from any tumor mass (× 300).

blood cells. The stroma supporting these vascular structures varies considerably—here and there the endothelial masses appear to be invaded by fibroblasts and strands of fibrous tissue whereas in other places the vascular structures are widely separated by dense bands of fibrous connective tissue. Wright[1] has pointed out the "dual tendency of endothelial cells in lymphoid structures to show epithelial qualities and the potentialities of connective tissue with reticulum formation." Certainly the microscopic picture of this neoplasm shows a resemblance toward both an endothelial structure and sarcoma. Another characteristic of angiosarcoma is the rapid rate of growth with invasion of surrounding structures and metastases to distant sites but more interesting is the observation that these tumors may be of multicentric origin. The finding of small vascular spaces (Figure 5) lined by large swollen and irregular endothelial cells within the otherwise uninvolved parenchyma of adjacent organs supports this contention.

Some of the reported cases have been associated with abdominal trauma. It has been observed[2] that in cases of artificial pneumoperitoneum there are secondary changes in the peritoneum (especially the visceral peritoneum) consisting of proliferation of mesothelium, blood vessels and fibrous connective tissue. While there is uncertainty as to the pathogenesis of angiosarcoma one cannot overlook the possibility that irritation of the peritoneal cavity such as that provoked by long standing artificial pneumoperitoneum might be a factor in the development of the tumor.

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Bilateral Pulmonary Resection for Metastatic Osteogenic Sarcoma: Case Report of a Seven Year "Cure"

W. R. RUMEL, M.D., F.C.C.P., P. R. CUTLER, M.D., F.C.C.P
and W. R. HALLIDAY, M.D.
Salt Lake City, Utah

The merit of excision of a solitary metastatic pulmonary malignancy in selected cases now seems well established, if the primary lesion can be controlled.[1-5] Many patients have been reported free of such disease for significant periods, one as long as 18 years after pulmonary resection.[6] However, there have been only a few reported attempts to eradicate bilateral pulmonary metastases.[2, 4, 5, 7-10] Nearly all of these have been cases with an apparently solitary metastasis with subsequent appearance of an additional metastatic lesion in the other lung. The only prolonged survival following resection of such bilateral metastatic pulmonary neoplasms, which could be found in a search of the American literature, was a case of a spindle cell neurosarcoma reported by Alexander.[11] No evidence or recurrence developed eight and one-half years after her second lobectomy and nine and one-half years after the discovery of the first pulmonary metastasis.

In view of the current low mortality and morbidity rates associated with pulmonary resection, it has been our belief that the discovery of multiple or bilateral pulmonary metastases should not alone preclude attempts at surgical cure if the total number and distribution of the metastatic lesions is reasonably favorable from a surgical standpoint. Review of the literature indicates that others also hold this view, since at least four such cases have been reported.[5, 8-10] In the first two of these, the second lobectomy was delayed six and eight months respectively. However, both patients soon died, one with extensive pulmonary metastases and the other with involvement of the liver. The third was free of disease six months following the second operation and the fourth had later undergone removal of pulmonary and renal recurrences with a subsequent follow-up of only five months.

Because of the limited overall experience in dealing with the excision of bilateral pulmonary metastases, a report of an additional case treated successfully seems justified.

From the Department of Thoracic Surgery, Latter-day Saints Hospital.

Case Report

In 1949 a local orthopedic surgeon* asked us to care for a 15 year old white girl who had multiple, bilateral pulmonary metastatic osteogenic sarcomata. In December, 1948, she had been hospitalized because of complaints referable to her right leg. Mild pain and a sensation of unusual warmth had been present in her knee for two months. Initial x-ray films of the joint were negative, but two weeks before admission when increased pain and swelling had developed, a rarified lesion in the distal end of the femur was demonstrated. Physical examination revealed no abnormality except for moderate swelling in this area. Laboratory studies were normal except that the alkaline phosphatase was 7.0 Bodansky units. A routine chest x-ray film was negative (Figure 1). On December 3, 1948, a mid-thigh amputation was performed, followed by an uneventful convalescence. The histologic diagnosis was osteogenic sarcoma. Routine x-ray films 11 months postoperatively revealed bilateral pulmonary metastases, although she was entirely asymptomatic (Figure 2). Physical and laboratory examinations were negative except for the evidence of amputation. On November 22, 1949, a right middle lobectomy was performed with removal of a solitary lesion measuring 5 cm. in diameter. On December 14, 1949, left lower lobectomy and lingulectomy with partial pericardiectomy was performed. The lower lobe contained two masses, each being 2 cm. in diameter and the lingula another, which was 4 cm. in diameter. Fibrous adhesions bound the lingular tumor to the pericardium in the region of the phrenic nerve but no gross invasion of the pericardium was present. After the second operation she developed a critical episode of hypotension with cardiovascular collapse and hyperthermia of 106°, followed by oliguria, but she recovered fully. The histological appearance of the pulmonary lesions was identical to that of the primary femoral tumor. Subsequently, she has been normally healthy and active in all respects, being able to graduate from the State University and to continue on with steady employment after graduation. No clinical or radiological evidence of recurrence had developed up until March 27, 1957, more than seven years after her second resection (Figure 3).

*Dr. A. M. Okelberry, L. D. S. Hospital, Salt Lake City, Utah.

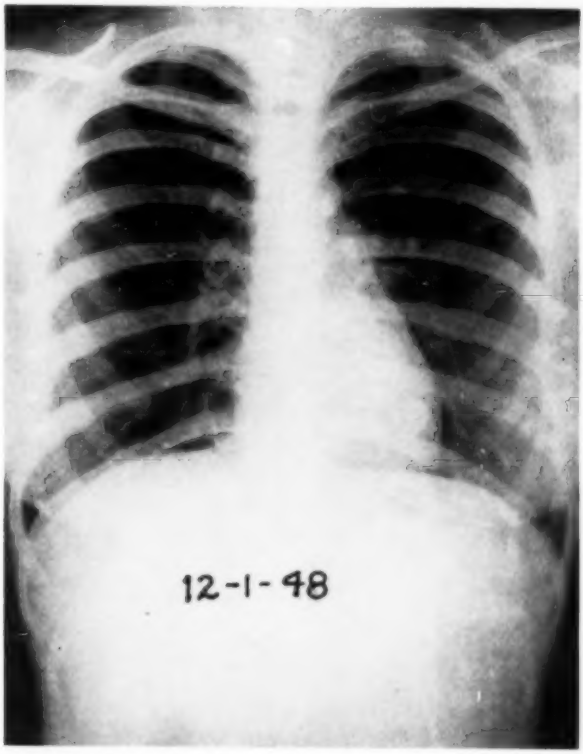

FIGURE 1: Normal roentgenogram before mid-thigh amputation.

DISCUSSION

In the majority of instances pulmonary metastasis must result from tumor invasion of the regional veins followed by embolization, with or without local trauma, If the lesion is solitary and resection results in "cure," there can be no reasonable doubt that the embolization involved a single cell or clump of cells breaking away on a single occasion. Obviously, if the primary growth is not completely resected, further embolic episodes may, and generally do, occur periodically resulting in further implants in new areas of the lungs. Logically there would seem to be no reason why multiple cells or clumps of cells might not be dislodged into the blood stream as an isolated occurrence or repeatedly over a short period of time, particularly if the primary lesion were traumatized. Still, if the distribution and total number of the implants happened to be favorable, from the standpoint of resectability, excision should be possible for the same reasons that "cure" of solitary metastases has been attained. Again, we must assume that the primary growth would have been eradicated completely. Admittedly, an increase in the total number of implants, especially if the distribution were widespread throughout both lungs, would be followed by a decrease in the likelihood of "cure." In the above case the variation in the size of the nodules strongly suggested that more than one and probably three episodes of tumor embolization took place before extirpation of the primary tumor. The appearance of multiple bilateral lesions up to 5 cm. in diameter within a year after resection of the primary growth would seem to indicate that the tumor was not one of unusually low-grade malignancy, but an ordinary osteogenic sarcoma. Also, the microscopic characteristics favored this contention. In spite of these considerations, which would detract from the likelihood of success, the result obtained appears to satisfy the usual criteria of a "seven year cure" following resection of multiple bilateral pulmonary metastases arising from a primary osteogenic sarcoma of the lower end

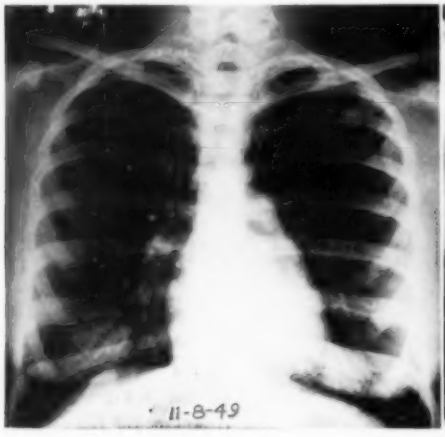

FIGURE 2

FIGURE 3

Figure 2: Bilateral metastatic osteogenic sarcomata 11 months after mid-thigh amputation.—*Figure 3:* Roentgenogram over 7 years after resection of bilateral metastatic osteogenic sarcomata—showing no recurrence.

of the femur, and indicates that the presence of such metastases does not necessarily preclude the possibility of "cure" following surgical excision.

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11. Alexander, J. (In discussion of Seiler et al'.)

AMENDMENT TO BYLAWS

The following amendment to the College Bylaws was adopted at the Semi-Annual Meeting of the Board of Regents on November 29, 1958 and will be presented to the membership for adoption at the annual Administrative Session in Atlantic City on Thursday, June 4, 1959, at the time of the Annual Meeting of the American College of Chest Physicians.

It is recommended that Article XII, Section 5: Council on International Affairs, (a), be amended as follows:

There shall be a Council on International Affairs, consisting of seven members to be appointed by the President of the College. The chairman of the council is to be appointed by the President to serve for a period of two years. The remaining six members are to be the chairmen of the following councils which serve under the Council on International Affairs: Council on Pan-American Affairs; Council on European Affairs; Council on Pan-Pacific Affairs; Council on African Affairs; Council on Asian Affairs; and Council on Middle Eastern Affairs.

The President of the College shall appoint at least one member from each country to serve on these six councils. The chairman, vice-chairman and secretary of each council shall be appointed by the President of the College for a period of two years.

Explanation

The original four councils were Pan-American, European, Pan-Pacific and African and Eastern Affairs. Due to the international growth of the College, it is deemed advisable to reorganize these councils to more adequately serve our purposes. It is therefore recommended that there be six councils organized to cover the 89 countries and territories where there are College members.

The reorganized councils, with the countries and territories they serve, are as follows:

Council on Pan-American Affairs

Argentina
Bolivia
Brazil
Canada
Canal Zone
Chile
Colombia
Costa Rica
Cuba
Dominican Republic
Ecuador
El Salvador
Guatemala
Haiti
Hawaii
Honduras
Jamaica
Martinique
Mexico
Nicaragua
Panama
Paraguay
Peru
Puerto Rico
Trinidad
United States
Uruguay
Venezuela
Virgin Islands

Council on European Affairs

Austria
Belgium
Czechoslovakia
Denmark
England
Finland
France
Germany
Greece
Hungary
Ireland
Italy
Netherlands
Norway
Poland
Portugal
Scotland
Spain
Sweden
Switzerland
Turkey
Wales
Yugoslavia

Council on African Affairs

Algeria
Belgian Congo
British East Africa
Liberia
Morocco
Portuguese East Africa
Portuguese West Africa
South Africa
Southern Rhodesia
Tunisia
West Nigeria

Council on Asian Affairs

Burma
Cambodia
Ceylon
China
India
Malaya
Nepal
Pakistan
Thailand
Vietnam

Council on Pan-Pacific Affairs

Australia
China (Republic of)
Hong Kong
Japan
Korea
Philippine Islands

Council on Middle Eastern Affairs

Bahrain
Egypt
Iran
Iraq
Israel
Jordan
Kuwait
Lebanon
Saudi Arabia
Syria

College Chapter News

PUERTO RICO CHAPTER

The Puerto Rico Chapter of the College held its annual meeting in Rio Piedras on November 19. A panel discussion on "Modern Trends in the Treatment of Pulmonary Tuberculosis" was presented in which Drs. Edmundo Figueras, David E. García, E. Martínez Rivera, José L. Porrata, Jaime F. Pou, Hilarión Sánchez and José E. Sifontes participated. Dr. Edward A. Gaensler, Boston, Massachusetts, was guest of honor at the meeting.

New Chapter Officers

COLORADO CHAPTER

President	James A. Wier, Denver
Vice President	Mordant E. Peck, Denver
Secretary-Treasurer	Leroy Elrick, Denver (re-elected)

GREEK CHAPTER

President	Nicolas Tsamboulas, Athens
Vice President	Sapho Mangriotis, Athens
Secretary-Treasurer	John Lameras, Athens (re-elected)

KENTUCKY CHAPTER

President	Walter P. Mayo, Lexington
Vice President	Oren A. Beatty, Louisville
Secretary-Treasurer	Daniel N. Pickar, Louisville

LOUISIANA CHAPTER

President	Morton M. Ziskind, New Orleans
1st Vice President	Howard A. Buechner, New Orleans
2nd Vice President	Dwight S. Danburg, Greenwell Springs
Secretary-Treasurer	William Leon, New Orleans

PACIFIC NORTHWEST CHAPTER

President	Kenneth A. Tyler, Gooding, Idaho
Vice President	Edward H. Morgan, Seattle, Washington
Secretary-Treasurer	Clyde Dean Packer, Blackfoot, Idaho

PENNSYLVANIA CHAPTER

President	David A. Cooper, Philadelphia
Vice President	John M. Snyder, Bethlehem
Secretary-Treasurer	Robert L. Mayock, Philadelphia (re-elected)

POTOMAC CHAPTER

President	William L. Cooke, Charleston, West Virginia
Vice President	Milton B. Kress, Baltimore, Maryland
Secretary-Treasurer	Joseph W. Peabody, Jr., Washington, D.C., (re-elected)

PUERTO RICO CHAPTER

President	José L. Porrata, Hato Rey
Vice President	Juan Ramón Marchand, Santurce
Secretary-Treasurer	Hilarión Sánchez, Rio Piedras

SOUTHERN CHAPTER

President	Daniel F. Jenkins, Houston, Texas
1st Vice President	John H. Seabury, New Orleans, Louisiana
2nd Vice President	DeWitt C. Daughtry, Miami, Florida
Secretary-Treasurer	Henry R. Hoskins, San Antonio, Texas

VIRGINIA CHAPTER

President	Samuel M. McDaniel, Norfolk
Vice President	Charles G. Pearson, Charlottesville
Secretary-Treasurer	George E. Ewart, Richmond

AWARDS TO BE MADE AT ANNUAL FILM SESSION

The Board of Regents of the College approved a recommendation proposed by the Committee on Motion Pictures to present awards for outstanding films presented at the 25th Annual Meeting in Atlantic City, June 3-7, 1959. Physicians having new films ready for presentation at the meeting are invited to submit all pertinent information to Dr. Paul H. Holinger, Chairman, Committee on Motion Pictures, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois. Prize-winning films will be announced at the Annual Presidents' Banquet of the College to be held at the Ambassador Hotel, Saturday night, June 6, 1959.

ANNOUNCEMENTS

In keeping with the celebration of our Silver Anniversary year, the Editorial Board and the Managing Editor's office have determined to revise the cover of *Diseases of the Chest*. It is hoped that our readers will find the new cover, initiated with this issue, a pleasing change.

Drs. Chevalier L. Jackson and Charles M. Norris have announced a Postgraduate Course on Bronchoesophagology will be given at the Department of Laryngology and Bronchoesophagology, Temple University Medical Center, Philadelphia, February 16-27, 1959. The tuition fee for the course is $250. Applications and further information may be obtained by writing to: Jackson-Research, Laboratory 604, Temple University Medical School, 3400 North Broad Street, Philadelphia 40, Pennsylvania.

A Symposium on Chest Diseases, sponsored by the Southwest Chapter of the Missouri Academy of General Practice, with the cooperation of the Missouri Chapters of the American Trudeau Society and the American College of Chest Physicians, and the Missouri State Sanatorium, will be presented at the Missouri State Sanatorium, Mt. Vernon, January 17 and 18.

Fellows of the College participating in the symposium are: Drs. Charles A. Brasher, W. W. Buckingham, Michael L. Furcolow, Harry V. Langeluttig, J. Arthur Myers, Karl H. Pfuetze, John W. Polk, Henry C. Sweany, and J. Lewis Yates.

Book Reviews

THE SALIENT POINTS & THE VALUE OF VENOUS ANGIOCARDIOGRAPHY IN THE DIAGNOSIS OF THE CYANOTIC TYPES OF CONGENITAL MALFORMATIONS OF THE HEART, by Benjamin M. Gasul, et al., Charles C Thomas, Springfield, 1957, $3.50

This little volume is a very valuable reference for those who would attempt to interpret the angiocardiogram.

The authors have recorded the results of a ten year study of 421 series of angiocardiograms done on patients with cyanotic types of congenital anomalies. Each has been carefully verified and checked against the various clinical and laboratory studies available.

Twenty-one separate entities were found in which cyanosis was present and these they have divided into 4 groups: Group 1, in which the diagnosis can *almost always* be made; Group 2, in which it can *usually* be made; Group 3, in which it can usually *not* be made, and Group 4, in which additional studies are always required.

The authors then proceed to outline the salient features of each group using line drawings and diagrams as well as the actual radiographic reproductions. The pathology, hemodynamics, and the diagrams of blood flow with the relative amounts of venous and arterial blood are all included for each entity.

As a ready reference for the clinician and the laboratory worker, this volume should have an enthusiastic reception. It provides an easy graphic review in cases which are always puzzling and challenging at best, in spite of all the data one may have at hand.

The volume has a nice format, and is a credit to both publisher and the authors.

Paul C. Swenson, M.D.

PERIPHERAL CIRCULATION IN HEALTH AND DISEASE by Walter Redisch, Francisco F. Tangco and R. L. deC. H. Saunders, Grune & Stratton, New York, 1957, $7.75

This small volume dealing with peripheral circulation in health and disease is essentially a concise summary of much of the literature published in this field in recent years.

Part I, covering the basic aspects of peripheral blood flow, includes a brief but accurate discussion of the anatomy and physiology of the vascular system. This part deals also with the evaluation of blood flow and numerous methods of examination are described. These tests will be of chief interest to the physiologists inasmuch as most of them have been discarded by the clinicians. It is unfortunate that such an obscure test as the Kisch test has been described and the well-known Allen test has been omitted.

Part II, dealing with pathologic alterations in peripheral blood flow, presents a somewhat confusing picture to the reader, especially from the standpoint of classification. Although the term obliterative arteriosclerosis is used by many, the commonly accepted terminology at the present time is arteriosclerosis obliterans. To be consistent, the authors should refer to thromboangiitis obliterans as obliterative thromboangiitis. One questions the accuracy of the statement that "Histopathologically, obliterative arteriosclerosis is characterized by medial sclerosis (Moenckeberg's arteriosclerosis) with varying degrees of intimal thickening as a result of superimposed atheromatosis or atherosclerosis." The current concept certainly does not include medial sclerosis as a basic characteristic of arteriosclerosis obliterans.

The section devoted to physiologic responses to disturbances in blood flow is well done and describes accurately our present knowledge of collateral circulation.

In a small volume of this type the essentials of management and therapy cannot be adequately described and the authors rightfully refer the reader to other textbooks. However, a great deal of space is devoted to various drugs, many of which are therapeutically ineffective.

Part V entitled "The Anatomic Basis of the Peripheral Circulation in Man" is extremely interesting and presents original investigative work. This section is the only one containing adequate illustrations and they are extremely well done.

A valuable contribution is the excellent documentation of references which will be of considerable help to the student in this field.

Fay A. LeFevre, M.D.

ENCYCLOPEDIA OF THORACIC SURGERY by E. Derra. Volume I, Springer-Verlag, Berlin, 1958, 838 pages, DM 370.

Professor E. Derra of Dusseldorf has undertaken the Herculean task of editing a three volume encyclopedia of thoracic surgery. By assigning to well known European and American scientists the various subjects in which they are eminently qualified, the editor has compiled this monumental work which, as judged by its first volume, should prove of tremendous value to all interested in diseases of the cardio-pulmonary systems. The Springer publishing firm should be complimented not only for having taken the initiative in bringing us this timely encyclopedia but also for having maintained their usual high caliber of craftsmanship.

The first volume, written by authorities in the fringe areas of thoracic surgery, deals with anatomy, physiology, and diagnostic methods as well as with anesthesia, hypothermia, and extracorporeal circulation. By far the outstanding portion is that on surgical anatomy of the thorax, prepared by Henry v. Hayek of the University of Vienna. Apparently a revision with additions of "Die Menschliche Lunge" which appeared in 1953, material of no particular interest to the surgeon, has been deleted and more emphasis has been placed on functional and surgical anatomy.

The normal and pathological physiology of respiration and circulation is an excellent review of what is generally known and accepted at the present time. A bibliography of 28 pages certainly indicates the thoroughness of this excellent, concise review prepared by Knipping, Bolt, Valentin, Venrath, Grosse-Brockhoff, and Schoedel.

The portion on general diagnostic methods by Dick discusses briefly the various standard procedures used in all well established thoracic clinics. The application of the diagnostic thorascopy has been re-emphasized, a method not in common use in this country. Diagnostic roentgenography, electrocardiography, angiocardiography, and heart catheterization are dealt with quite adequately by Vieten, Bayer, Werko, and Kjellberg. No mention is made of left heart catheterization,

although this procedure is replacing right heart catheterization for the investigation of patients with acquired heart disease.

The chapter on anesthesia is altogether too brief and gives very little factual information. There is an excellent discussion of the physiological alterations produced by and clinical applications of hypothermia by Zindler. Since this method of open heart surgery is being rapidly abandoned, this portion of the encyclopedia will have limited value in the future.

The rapid technical advances and mounting clinical experiences in extracorporeal circulation of the past 18 months could not be included in Salisbury's excellent review of this subject and indicate the impossibility of keeping this encyclopedia up to date at this stage of the development of cardiac surgery.

The second and third volumes, scheduled for publication this year, deal with the surgery of the lung and heart respectively. The list of contributors is a Who's Who in thoracic surgery. Early revision of volumes I and II is mandatory to prevent this work from becoming an historical rather than practical reference book.

The *Encyclopedia of Thoracic Surgery* is a well planned and beautifully executed work which does credit to the German scientists and publishing firm. This encyclopedia is a must for all university and hospital libraries and even though of necessity quite costly, should be available to all in the field of cardio-pulmonary diseases. The major portion of this first volume is written in German, a factor which unfortunately may limit its demand in this country.

Karl P. Klassen, M.D.

Obituaries

AFFONSO MAC DOWELL
1881-1958

Affonso Mac Dowell, one of the outstanding Brazilian phthisiologists, who died in Rio de Janeiro on April 13, 1958, at the age of 76, was born at Belem in 1881, and graduated from the Rio de Janeiro Medical School in 1904.

The first years of his professional life were dedicated to the study and treatment of tropical diseases in his native town, Belem, where his research work resulted in publication of many original and interesting papers. Later, established in Rio de Janeiro, he became Associate Professor at the Rio de Janeiro Medical School and in 1929, became Director of the Tuberculosis Service of the General Policlinic. At this time, he founded the *Brazilian Review of Tuberculosis*. His numerous publications on experimental and clinical tuberculosis revealed the scientist as well as the experienced practitioner. In recognition of his major contributions, chiefly in the field of tuberculosis, he has been awarded membership in many Brazilian and foreign scientific Associations, among these the National Academy of Medicine, the Brazilian Tuberculosis Society and the American College of Chest Physicians. He was President of the Brazilian Tuberculosis Society, the 1st Brazilian Congress on Tuberculosis in 1938, the Federation of Brazilian Tuberculosis Societies, and Governor of the Brazilian Chapter of the American College of Chest Physicians. Due to his remarkable work in the field of tuberculosis, he was awarded the "Medal Cardoso Fontes" by the Brazilian Tuberculosis Society.

Surviving are two sons, Affonso Mac Dowell Filho and Samuel A. Mac Dowell, both phthisiologists, as well as four daughters and many grandchildren.

Mauricio Teichholz, M.D.
Governor for Rio de Janeiro

FRANK I. TERRILL
1901-1957

Dr. Frank I. Terrill died of a coronary occlusion on December 21, 1957, at the age of 56. He was Superintendent of the Montana State Tuberculosis Sanitarium at the time of his death, a position he had filled for twenty years.

Dr. Terrill was recognized as a pioneer in the Pacific Northwest in the fields of thoracic surgery, bronchoesophagology and the treatment of tuberculosis. He was graduated in 1925 from the University of Michigan Medical School, and did

postgraduate work in Vienna, Austria, and at leading medical centers in the United States.

He was a Fellow of the American College of Chest Physicians and on its Board of Governors for many years. He was a past-president of the Pacific-Northwest Chapter of the American College of Chest Physicians.

Dr. Terrill was a Fellow of the American College of Surgeons and a member of the American Association for Thoracic Surgery. He was also a member of the National Trudeau Society, the first president of the Montana Trudeau Society, and belonged to the National Tuberculosis Association.

He is survived by his wife, Audrey, who resides in Anaconda, Montana.

Dominic M. Ferrando, M.D.

FELIX BAUM
1883-1958

Dr. Felix Baum of South Orange, New Jersey, died at his home of coronary thrombosis at the age of 74. He was born in Germany, and after attending the University of Heidelberg, received his medical degree from Leipzig University. He came to the United States in 1924 and served as Medical Director of the National Jewish Hospital in Denver, Colorado. He was also an Associate Professor of Medicine at the University of Colorado. He came to Newark in 1927 and began private practice in the specialty of chest diseases. He moved his practice to South Orange, New Jersey in 1940. He was a pioneer in the field of thoracic surgery.

Dr. Baum was one of the organizing members of the New Jersey Chapter of the American College of Chest Physicians. He is the father of two other Fellows of the College, Dr. Otto Baum and Dr. Lewis Baum now president-elect of the New Jersey Chapter.

For many years, he was an attending physician at the Deborah Sanatorium, Browns Mills, New Jersey, and also consultant physician at St. Mary's Hospital, Orange, New Jersey, and the Essex County Sanatorium, Verona, New Jersey.

Dr. Baum was a member of the American Medical Association, the New Jersey State Medical Society and the Essex County Medical Society. He was also a member of the Academy of Medicine of New Jersey, the American Academy of Tuberculosis Physicians and the American Trudeau Society.

His demise has taken an active, sincere pioneer in the field of pulmonary pathology. His friends will long remember him for his interest in the field of surgery, and for his sincere efforts to promote progress in the field of pulmonary disease.

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June 3-7, 1959

Homecoming Meeting
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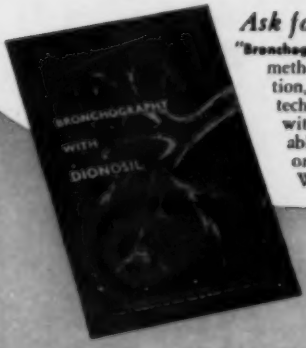

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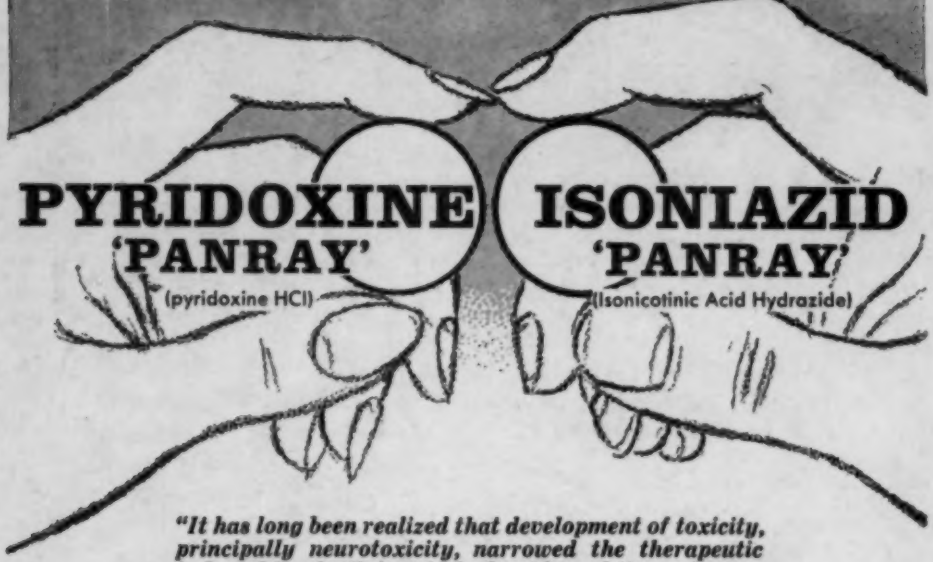

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[1]Sea View Bulletin 16:52 July '56 Tchertkoff, et al. JAMA 156:1549 (Dec. 25) 1954 Biehl & Viltner
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